REMEDIAL INVESTIGATION AND FEASIBILITY STUDY FOR THE SOMERS TIE PLANT

Volume II: Exposure & Endangerment Assessment

Prepared for
Burlington Northern Railroad
Seattle, Washington

Prepared by
Remediation Technologies, Inc.
Kent, Washington

September 1988

TABLE OF CONTENTS

Volume I: Site Investigation

Section			Page
1. Introduction			1-1
	1.1	Purpose	1-1
	1.2	Site Background Information	1-2
		1.2.1 Site Description	1-2
		1.2.2 Site History	1-2
		1.2.3 Site Investigations	1-5
		1.2.4 Previous Waste Removal Activities	1-6
	1.3	Overview of Report	1-10
2. Site Features Investigation			2-1
	2.1	Demography	2-1
	2.2	Land Use	2-1
	2.3	Climatology	2-3
	2.4	Natural Resources	2-5
	2.5	Cultural Resources	2-7
		2.5.1 Prehistoric Resources	2-7
		2.5.2 Historic Resources	2-7
3. Hazardous Substance Investigation			3-1
	3.1	Treatment Operations	3-1
		3.1.1 Zinc Chloride Treatment	3-1
		3.1.2 Creosote Treatment	3-2
	3.2	Waste Generation and Disposal	3-4
		3.2.1 Wastewater	3-4
		3.2.2 Spills and Drippage	3-5
		3.2.3 Sludges	3-8
	3.3	Waste Sampling	3-8
		3.3.1 HSL Organics	3-11
		3.3.2 HSL Inorganics	3-14
		3.3.4 Summary and Selection of Chemicals-of-Interest	3-16
	3.4	Characteristics and Behavior of Wood Preservatives	3-16
		3.4.1 Creosote	3-16
		3.4.1.1 Fate of PAH	3-17
		3.4.1.2 Toxicity of PAH	3-19
		3.4.2 Zinc Chloride and Chromated Zinc Chloride	3-22
		3.4.2.1 Fate of Zinc	3-22
		3.4.2.2 Toxicity of Zinc	3-23

3.4.2.3 Fate of Chromium 3-24
3.4.2.4 Chromium Toxicity 3-24

4. Soil and Geologic Investigations 4-1

4.1 Regional Soils Data 4-1
4.2 Soil Sampling and Analysis Procedures 4-1
4.2.1 Field Sampling 4-1
4.2.1.1 Borings 4-2
4.2.1.2 Test Pits 4-5
4.2.1.3 Background Soil Sampling 4-8

4.2.2 Laboratory Analysis 4-8

4.3 Soil Quality Data 4-8
4.3.1 Organic Results 4-8
4.3.2 Inorganic Results 4-14
4.3.3 Summary of Soil Investigations 4-14
4.3.3.1 CERCLA Lagoon 4-15
4.3.3.2 Ditch 4-16
4.3.3.3 Drip Track 4-17
4.3.3.4 Swamp Area 4-17
4.3.3.5 Tie Storage 4-18

4.4 Geology 4-18
4.4.1 Regional Geology 4-18
4.4.1.1 Precambrian Rocks 4-18
4.4.1.2 Quaternary Deposits 4-19
4.4.1.3 Structure 4-21
4.4.1.5 Glaciation 4-21

4.4.2 Local Geology 4-22

5. Hydrogeologic Investigations 5-1

5.1 Regional Hydrogeologic Conditions 5-1
5.1.1 Precambrian Bedrock Aquifer 5-1
5.1.2 Pleistocene Aquifers 5-2
5.1.3 Recent Alluvium Water Table Aquifers . . 5-3

5.2 Local Hydrogeology 5-4
5.3 Groundwater Sampling and Analysis Procedures . . 5-20
5.3.1 Field Sampling 5-20
5.3.2 Laboratory Analysis 5-22

5.4 Groundwater Quality Results 5-22
5.4.1 Organic Results 5-27
5.4.2 Inorganic Results 5-28
5.4.3 Summary of Groundwater Quality Results . 5-31
5.4.3.1 CERCLA Lagoon Area 5-31
5.4.3.2 Swamp Area 5-33
5.4.3.3 RCRA Impoundments 5-33

5.5 Groundwater Modeling 5-34
5.5.1 Model Description 5-34
5.5.3 Model Sensitivity 5-37
5.5.3.1 Hydraulic Conductivity 5-37
5.5.3.2 Dispersivity 5-37
5.5.3.3 Biodegradation of the Solute . . 5-37

5.5.4 Summary of Modeling 5-38

6. Surface Water and Sediment Investigations 1

6.1 Literature Review and Background Data - Flathead Lake 1
6.1.1 Hydrodynamics 3
6.1.2 Trophic State 4
6.1.3 Water Quality Classification 4

6.2 Literature Review and Background Data - Somers Slough 6
6.3 Surface Water Quality Sampling and Analysis Procedures 6
6.3.1 Sampling Procedures - Near Shore Surface Water 7
6.3.2 Flathead Lake Sampling 7
6.3.4 Laboratory Analysis Procedures 9

6.4 Analytical Results 9
6.4.1 Flathead Lake - Organics 9
6.4.2 Flathead Lake - Inorganics 13
6.4.3 Slough 15
6.4.4 Summary 17

6.5 Sediments Sampling and Analysis Procedures . . 18
6.5.1 Sampling Procedures 18
6.5.1.1 Lake Sediments 18
6.5.1.2 Beach Borings 18
6.5.1.3 WPA Sediments 22
6.5.1.4 Slough Sediments 22

6.5.2 Laboratory Analysis 24

6.6 Sediment Sampling Results 24
6.6.1 Organics 25
6.6.2 Inorganics 25
6.6.3 Summary 28

7. Air Quality Investigation 1

7.1 Workplace Monitoring 1
7.1.1 Sampling and Analysis Methods 1
7.2.1 Measured Air Quality Concentrations 2

7.2 Ambient Modeling 6
7.2.1 Modeling Method 6
7.2.2 Air Quality Impact Analysis 9

7.3 Modeling Verification and Sensitivity 11
7.3.1 Analysis Methods 11
7.3.2 Results 14

8. Drinking Water Investigations 1

8.1 Description of Private Wells 1
8.2 Field Sampling 3
8.3 Laboratory Analysis 4
8.4 Private Well Results 4
8.5 Municipal Water Supply Results 7
8.5.1 Inorganic Results 7
8.5.2 PAH Results 7

8.6 Data Discussion 10
8.7 Summary 12

9. Ecological Investigations 1

9.1 Sampling of Aquatic Life 1
9.1.1 Sample Collection 1
9.1.2 Aquatic Life Sampling Results 4

9.2 Waterfowl Observation and Sampling 6
9.2.1 Nest Searching 8
9.2.2 Waterfowl Observations 9
9.2.3 Waterfowl Sampling 10
9.2.3.1 Methods 10
9.2.3.2 Sample Collection 10
9.2.3.3 Sample Preparation 11
9.2.3.4 Sample Analysis 11

9.2.4 Summary 12

10. Pilot and Bench Scale Studies 1

10.1 Groundwater Extraction 1
10.2 Groundwater Treatment 1
10.3 Incineration 3
10.4 Land Treatment 12
10.4.1 Test Plot Construction 15
10.4.2 Sampling and Analysis 16
10.4.3 Results and Data Analysis 17

Volume II: Exposure & Endangerment Assessment

Section		Page
11.	Exposure and Risk Assessment	1
11.1	Exposure from Domestic Water Supplies	3
11.1.1	Precambrian Bedrock Aquifer	3
11.1.2	Lower Artesian Aquifer	5
11.1.3	Deltaic Sand Aquifer	6
11.1.4	Surface Water Supplies	8
11.2	Exposure of Residents to Contaminants in Air	10
11.3	Exposure From Ingestion of Fish	10
11.4	Exposure by Direct Contact with Soils/Sediments	14
11.5	Exposure by Direct Contact While Swimming	17
11.6	Public Heath Risk Assessment	19
11.6.1	Risks Associated with Drinking Water	21
11.6.1.1	Private Wells	21
11.6.1.2	Municipal Supply	23
11.6.1.3	Hypothetical New Well	25
11.6.2	Risks Associated with Exposure to PAH Compounds in Air	25
11.6.3	Risks Associated with Exposure to Soils/Sediments	27
11.6.4	Risks Related to Ingestion of Fish	27
11.7	Summary of Risks	29
12.	Environmental Exposures	1
12.1	Plant Uptake of PAH	1
12.2	Cattle Uptake of PAH	2
12.2.1	Literature Review	2
12.2.1	Potential for Cattle Uptake of PAH at Somers	4
12.3	Aquatic Life Uptake of PAH and Zinc	6
12.3.1	Literature Review of PAH Uptake	6
12.3.2	Literature Review of Zinc Uptake	8
12.3.2	Sampling of Aquatic Life	11
12.4	Waterfowl Uptake of PAH	12
12.4.1	Literature Data on PAH Uptake by Waterfowl	12
12.4.2	Potential for PAH Uptake by Waterfowl at Somers	14
12.5	Zinc Uptake by Waterfowl	17
12.5.1	Literature Review	17
12.5.2	Potential for Zinc Uptake by Waterfowl at Somers	20

12.5.3 Waterfowl Observation and Sampling 21

13. Risk-Based Clean-Up Goals for Somers 1

13.1 Noncarcinogenic Compounds 1
13.2 Carcinogenic Compounds 3

Volume III: Feasibility Study

not included

Volume IV: Appendices A through F

Appendix A Aerial Photographs
Appendix B Waste Sampling Data
Appendix C Well, Boring and Test Pit Logs
Appendix D Soil Sampling Data
Appendix E Bailer Recover Test Data
Appendix F Groundwater Sampling Data

Volume V: Appendices G through M

Appendix G Groundwater Modeling Data
Appendix H Surface Water Sampling Data
Appendix I Sediment Sampling Data
Appendix J Air Quality Data
Appendix K Drinking Water Sampling Data
Appendix L Ecological Sampling Data
Appendix M Incineration Test Burn Data

LIST OF TABLES

Table		Page
1-1	Schedule of RI Activities	1-7
2-1	1980 Census Population of Somers	2-2
3-1	Summary of Organic Analysis - HSL Samples	3-13
3-2	Inorganic Analysis of HSL Samples	3-15
3-3	Physical/Chemical Properties of Selected PAH Compounds	3-18
3-4	Water Quality Standards and Criteria for Wood Preservatives Used at Somers	3-21
4-1	Soil Organic Data	4-9
4-2	Soil Inorganic Data	4-11
4-3	Well Data	4-23
4-4	Grain Size Data	4-31
5-1	Well Data	5-6
5-2	1986 Groundwater Elevations	5-7
5-3	1987 and 1988 Groundwater Elevations	5-8
5-4	Aquifer Test Data	5-19
5-5	Groundwater Quality Data - Organics	5-23
5-6	Groundwater Quality Data - Inorganics	5-24
6-1	Results of PAH Analysis of Flathead Lake Samples	6-12
6-2	Results of Inorganic Analysis Flathead Lake Samples	6-14
6-3	Slough Water Quality Data	6-16
6-4	Sediment Organics	6-26
6-5	Sediment Inorganics	6-27
7-1	OSHA Sampling Results	7-3
7-2	MDHES Sampling Results	7-4
7-3	Saturation Vapor Pressures and Emission Rates of PAH	7-8
7-4	Air Quality Modeling Results	7-10
7-5	Predictd Air Quality of Naphthalene Somers CERCLA Lagoon	7-15
8-1	Results of Private Well Sampling in Somers	8-5
8-2	Results of Municipal Water Supply Sampling in Somers	8-8
8-3	PPT PAH Analysis of Blanks	8-11
9-1	Dolly Varden - Lake Whitefish Samples	9-5
10-1	Pilot Unit Operating Conditions	10-5
10-2	Thermogravimetric Analyzer (TGA) Conditions	10-5
10-3	Semivolatile Compounds Determined in Waste Feed Samples	10-7
10-4	Semivolatile Compounds Determined in Incinerator Ash Samples - Pilot Scale Tests	10-9
10-5	Semivolatile Compounds Determined in Incinerator Ash Samples - TGA Tests	10-11
10-6	Semivolatile Compounds Determined in Scrubber Effluent Sample	10-12
10-7	Semivolatile Compounds Determined in Flue Gas Samples	10-13
10-8	Destruction and Removal Efficiencies of Hazardous Constituents	10-14
10-9	Average PAH Concentration in Paradise Land Treatment Test Plots	10-19

10-10 Paradise Land Treatment Test Plots Contaminant Half Lives Based on First Order Reaction Kinetics 10-20
10-11 Paradise Land Treatment Test Plots 10-21
11-1 Estimate of Potential Exposure to Drinking Contaminated Groundwater 11-9
11-2 Estimates of Exposures from Drinking the Somers Municipal Supply Water 11-11
11-3 Summary of Surface and Subsurface Soil Concentrations 11-15
11-4 Estimated Exposures from Ingestion of Soils. 11-18
11-5 Estimated AIC Values for Selected PAH Compounds using RFD Approach 11-20
11-6 Summary of Acceptable Intake Values and Carcinogenic Potency Factors 11-22
11-7 Assessment of Exposure Risk from Municipal Supply Water 11-24
11-8 Assessment of Exposure Risk from Groundwater 11-26
11-9 Estimate of Exposure Risk from Soil Ingestion 11-28
13-1 Assessment of Clean-up Goals for Noncarcinogenic Compounds 13-2
13-2 Assessment of Clean-up Goals for Carcinogenic Compounds 13-4

LIST OF FIGURES

Figure		Page
1-1	Site Location Map	1-3
1-2	Layout of the Somers Tie Plant	1-4
1-3	Schematic of 1988 Emergency Removal Action	1-9
2-1	Area Temperature and Precipitation Data	2-4
2-2	Flood Plain Map	2-6
3-1	Location of Disposal Lagoons	3-6
3-2	Location of Treated Tie Storage Areas	3-7
3-3	Tie Storage in Slough	3-9
3-4	Location of Hazardous Sbustance List Samples	3-12
4-1	Location of Wells and Borings	4-3
4-2	Test Pit and Monitor Well Locations near CERCLA Lagoon	4-6
4-3	Swamp Area Test Pits	4-7
4-4	Cross-Section Locations	4-24
4-5	Cross-Section A-A′	4-25
4-6	Cross-Section B-B′	4-26
4-7	Cross-Section C-C′	4-27
4-8	Cross-Section D-D′	4-28
4-9	Cross-Section E-E′	4-29
5-1	Monitor Well Locations	5-6
5-2	Ground Water Contours April 1986	5-11
5-3	Ground Water Contours July 1986	5-12
5-4	Potentiometric Surface Map Oct/Nov	5-13
5-5	Potentiometric Surface Map March	5-14
5-6	Potentiometric Surface Map June	5-15
5-7	Potentiometric Surface Map October	5-16
5-8	Potentiometric Surface Map December	5-17
5-9	Potentiometric Surface Map June 1988	5-18
5-10	TSS vs Zinc	5-30
5-11	Inferred Extent of Groundwater Contamination	5-32
6-1	Flathead River System to Flathead Lake	6-2
6-2	Temperature Profiles at Surface Water Monitoring Sites, July 1984	6-5
6-3	Flathead Lake Sampling Locations	6-10
6-4	Beach Boring Locations Along Sommers Beach	6-20
6-5	Phase II Sediment Location	6-21
6-6	Phase III Sediment Locations	6-23
8-1	Drinking Water Sampling Locations	8-2
9-1	Location of Gill Nets in Flathead Lake	9-2
9-2	Location of Slough Study Areas	9-7
10-1	Location of Test Groundwater Extraction Trench	10-2

11. EXPOSURE AND RISK ASSESSMENT

The objectives of the Exposure and Risk Assessment for the Somers site are to:

1. Identify actual or potential routes of contaminant exposure

2. Determine the extent of exposure and/or exposure probabilities

3. Quantify the current or potential risks associated with these exposures

An assessment of potential exposure pathways was conducted to determine which pathways might be significant for the site in terms of potential human and environmental exposure. The current extent of contamination on and around the site and the results of the fate and transport assessment for predicting future contamination migration pathways were examined. All existing routes were examined as well as those that may be expected in the future if no remedial measures are implemented at the site (worst case). Therefore, the exposure scenarios reflect conservative assumptions to ensure that risk characterization and criteria development are protective of all populations potentially at risk.

The primary routes of exposure have been identified for the Somers site based on the pathway screening analysis. They are:

Human Health

1. Exposure of residents by ingestion of contaminated water.

2. Exposure of residents to contaminants in ambient air.

3. Exposure by ingestion of fish caught in Flathead Lake.

4. Exposure by direct contact with soils and sediments at the site.

5. Exposure by direct contact with surface water while swimming.

Environmental

1. Exposure of wildlife on the site to contaminated soils and water.

2. Exposure of fish and other aquatic life to contaminated soils and water.

3. Exposure of grazing animals by ingestion of contaminated soils and vegetation.

Public health exposure assessments present a calculated intake or dose associated with a certain activity. This dose is determined by combining the concentration of a chemical in an environmental media (e.g. in air, water or soils) with the estimated intake of that media (e.g. the amount of air breathed, water drunk or soil ingested) to calculate the amount of chemical a person may be exposed to. Doses may be presented in terms of either milligrams of compound per day (mg/day) or milligrams of compound per day per kilogram of body weight (mg/kg/day). Doses can be further qualified as the average daily dose on a single day during which exposure occurs, the average daily dose over some selected time interval or averaging period, or the average daily dose over a lifetime.

Risks associated with the calculated exposures can then be estimated. Health effects data for certain chemicals have been developed by EPA and by other private or public organizations. These data are typically based on studies with laboratory animals. For known or suspected cancer causing compounds, carcinogenic potency factors (CPFs) are used. The development of these factors is based on the assumptions that 1) there is no threshold concentration below which cancer would not occur, i.e. any amount of the compound poses some risk; and 2) the relationship between concentration and effect is linear, i.e. doubling the concentration doubles the cancer risk. CPFs are obtained from the slope of the dose-response curve and are expressed in terms of $mg/kg/day^{-1}$.

Health effects associated with noncarcinogenic compounds can

be acute, subchronic responses or chronic (long-term) responses. Acute effects would result from exposures lasting hours or days and could result from direct contact with highly contaminated materials. Subchronic effects are associated with exposures of weeks to months while chronic effects are associated with long term exposures on the order of years. In most risk assessments, emphasis is placed on potential chronic health effects. These effects are evaluated with reference to acceptable intake chronic (AIC) values. AIC values serve as benchmarks and represent the exposure that could occur with no expected health effect. AIC values are in terms of mg/kg/day.

This section of the Somers RI/FS presents the public health exposure and risk assessment for current and potential future conditions. Section 12 presents data on the potential environmental exposures and provides an endangerment assessment. Data on recommended clean-up goals for the site based on these risk assessments are presented in Section 13.

11.1 Exposure from Domestic Water Supplies

Domestic water supplies are or could be drawn from three groundwater aquifers as well as from Flathead Lake. The potential for exposure from these supplies is discussed below. For each case, the exposure assessment makes the following assumptions:

1. Individuals drink two liters of water per day (adult consumption) over a lifetime of 70 years;

2. The average individual body weight is 70 kg; this weight is typically used for adults;

3. The concentrations of contaminants measured in the water is constant over the time period considered (i.e., there may be short-term temporal fluctuations but the average stays the same).

11.1.1 Precambrian Bedrock Aquifer

Section 5 of the Somers RI/FS described the characteristics of the Precambrian bedrock aquifer. Konizeski (1968) identified

wells drilled into bedrock along the east and west shores of Flathead Lake at Big Fork and Somers and southwest of Kalispell where this aquifer yields water for domestic use; however, he concluded that it is not a source of large groundwater supplies.

Two bedrock wells were sampled during the Somers RI. These are the wells at the school and at the Hersman residence. Only the school well is currently used as a source of drinking water. Sampling was conducted during February and November 1986 as described in Section 8. All metals were within drinking water standards. Three PAH compounds were detected in the two bedrock well. Results from the five samples analyzed in ng/l and the resultant exposures (mg/kg/day) are summarized below.

	Geometric Mean		Maximum Observed	
	(ng/l)	(mg/kg/day)	(ng/l)	(mg/kg/day)
Naphthalene	2.1	6.0x10-8	40	1.1x10-6
Acenaphthalene	1.8	5.1x10-8	7.6	2.2x10-7
Benzofluoranthenes	1.35	3.9x10-8	3.5	1.0x10-7

The potential for contamination of bedrock aquifers depends, in part, on the proximity of contamination to bedrock, on the transmissivity of sediments, and on local hydraulic conditions. Konizeski mapped the contact between the Precambrian bedrock forming the Salish Mountains and the Quaternary sediments of the Kalispell Valley along the western property line of the plant site. Approximately 200 feet east of the contact is a rock out crop located on the west side of the RCRA impoundments and the Sanitary Lagoon. A second outcrop forms the ridge west of the swamp. It is believed that these outcrops consist of Precambrian Bedrock and are connected by a bedrock ridge buried at shallow depth beneath the surface of this area.

Bedrock was not encountered on the site in the vicinity of the RCRA impoundments to a depth of 101 feet (S-85-4c). Bedrock has been cored in one boring located at the north end of the plant site (S-1). The drill log of boring S-85-7 notes "black shale ..., chunks of red, dark green shale, limestone, subangular

rounded, assumed bedrock" at depth 72 feet. It is possible that bedrock was encountered in this boring or that a boulder within the glacial sediments was encountered. Refusal to drilling was encountered in Boring S-85-8b at 99 feet and was assumed to be bedrock. Again the top of bedrock or a boulder may have been encountered. Well S-7 encountered an obstruction at depth 22.5 feet through which the boring could not be advanced. It is possible that this obstruction is part of the bedrock ridge discussed above or a boulder within the clayey sand deposit.

The potential for migration from contaminated area to the bedrock aquifer is thought to be limited. Due to the presence of an artesian aquifer system beneath the shallow water table aquifer, downward migration of contaminants to bedrock is not expected. However, the irregularity of bedrock at the site has been noted and the possibility that bedrock might be located just beneath surficial sediments in some areas of the site cannot be discounted entirely as a potential pathway for migration. Consideration must also be given to the fact that the town of Somers plans to switch from Flathead Lake to groundwater from the bedrock aquifer as their source of municipal water supply.

Given the nature of variability that has been observed in low level PAH analysis, the existing data do not provide a basis for concluding that contamination at the site has affected the bedrock aquifer system. Still, the risks associated with ingestion of drinking water containing the levels of PAH compounds discussed above will be examined.

11.1.2 Lower Artesian Aquifer

The Lower Artesian aquifer as described by Konizeski occurs throughout most of the Kalispell Valley and represents a significant source of groundwater for domestic supplies. This aquifer consists of a series of sand and gravel deposits separated by discontinuous beds of fine grained material. The thickness of the aquifer is generally unknown as most wells have penetrated only the top few feet. However, in one location, a well pene-

trated the aquifer for at least 364 feet.

In the vicinity of Flathead Lake, 38 wells penetrate the Lower Artesian Aquifer and range in depth from 125 feet to 480 feet and averaging 230 feet. The 38 wells are located primarily in the north and northeast of the site and are within the Flathead River valley.

Movement of groundwater within the upper (sand) aquifer is restricted due to the low hydraulic conductivity values found within this aquifer. Leakage to the lower artesian aquifer from the sand aquifer would be impeded due to artesian flow which would restrict downward contaminant movement. Thus, from the perspective of hydraulic conductivity, contamination of the main artesian aquifer would appear unlikely. Further, based on information summarized by Konizeski, the pumping wells in the Flathead Lake area appear to be upgradient from the site.

The Lower Artesian Aquifer has been penetrated at the Somers site in wells S-85-1b, S-85-6b and S-85-8b, as evidenced by artesian flow and/or upward gradients compared to other wells in the well group. Samples from these three wells were analyzed on several occasions for metals and PAH. No PAH were detected in any of the samples from these wells. The levels of lead in wells S-85-1b and S-85-6b exceed the primary drinking water standard for lead. In the absence of detectable PAH, however, it appears that negligible exposure of the artesian aquifer to contamination from the site has occurred. Yet, because creosote materials have been found at depth beneath the CERCLA lagoon, there is a small potential that some contamination might eventually reach the lower artesian aquifer. Quantification of the potential for contamination or of the likely magnitude of the contamination is not feasible with any degree of accuracy. A worst case scenario would be that the lower artesian aquifer would be of the same concentrations as the shallow sand aquifer.

11.1.3 Deltaic Sand Aquifer

The deltaic sand aquifer is located through out the Somers

area and consists of fine sand and silt. A discussion of local groundwater hydrology for the deltaic sand aquifer is presented in Section 5.

A well survey conducted as part of the Somers investigation revealed a limited number of shallow water table wells in the vicinity of the site. These were shown on Figure 8-1 and are all located north and upgradient of the site in terms of the flow of the shallow water table aquifer. Three of the locations are now on the Somers municipal supply and no longer use their wells.

Sampling was conducted on three of the private sand aquifer wells February 1986 and November 1986. All levels of metals in these wells were within drinking water standards. PAH compounds detected in the five samples included naphthalene, fluorene and benzofluoranthenes at the concentrations (ng/l) and calculated exposures (mg/kg/day) presented below:

	Geometric Mean		Maximum Observed	
	(ng/l)	(mg/kg/day)	(ng/l)	(mg/kg/day)
Naphthalene	4.4	1.3x10-7	40	1.1x10-6
Fluorene	1.15	3.3x10-8	2	5.7x10-8
Benzofluoranthenes	1.0	2.9x10-8	1	2.9x10-8

The numbers given above are considered in the risk assessment but it is judged unlikely that these shallow wells were impacted by contaminated groundwater flowing from the site.

The majority of the groundwater monitoring wells installed during the Somers RI provide data on the quality of the deltaic sand aquifer. Wells located within the CERCLA lagoon and adjacent to the former swamp pond contain significant levels of contamination and free oil has been noted in some (See Section 5.4). Future use of the groundwater within the source areas can be restricted by BN as they are the property owner. Three wells located off of BN property immediately downgradient from the CERCLA lagoon contained PAH and phenolic compounds. Data are available from the June and July 1988 sampling rounds. Total PAH concentrations in Wells S-88-1, 88-2 and 88-3 were 240 to 491

ug/l, 459 to 1962 ug/l and 1330 to 150 ug/l, respectively. No high molecular weight PAH were detected. Phenolic compounds, primarily phenol and methyphenols, totaled 197 to 206 mg/l, 21 to 12.6 mg/l and 0.8 to 1.0 mg/l respectively.

As these wells are not located on BN property, an assessment of the exposure from a potential new well installed there must be considered. Table 11-1 presents the parameters detected in wells S-88-1, 88-2 and 88-3 and calculates exposures related to drinking that water.

11.1.4 Surface Water Supplies

Figure 8-1 showed the location of the site with reference to the intake for the municipal water supply which is currently drawn from Flathead Lake. Chemicals introduced to the lake at the site have the potential for contaminating the drinking water.

Water sampling of the lake water and of the municipal water supply has been conducted to assess the current influence of the site on local surface water levels of PAH in Flathead Lake. Surface water sampling in Flathead Lake is described in Section 6. The results of municipal water supply sampling at Somers are presented in Section 8. The levels of metals in Flathead Lake and the municipal supply system were all within EPA drinking water standards.

Flathead Lake samples from April 1987 showed detectable PAH at three stations. One sample (Station E) contained naphthalene at 47 ng/l and acenaphthalene at 3.9 ng/l. In duplicate samples collected from another station (Station D), phenanthrene/anthracene was reported present at 6.1 ng/l in one of the samples and fluoranthene was reported present at 10 ng/l in the other sample. No high molecular weight PAH were detected in any of the samples.

There is the potential that increased quantities of PAH compounds may be released to the lake in the future via runoff, discharge of contaminated groundwater from around the swamp pond, and possible leaching of non-aqueous phase liquids (creosote) from the swamp/beach area. The magnitude of these sources or

Table 11-1

ESTIMATE OF POTENTIAL EXPOSURE

TO DRINKING CONTAMINATED GROUNDWATER

	S-88-1,2,3 Well Data			
	Geometric Mean (ug/l)	Maximum (ug/l)	Exposure (mg/kg/day) Geo. Mean	Maximum
PHENOL	271.2	31000	7.7E-03	8.9E-01
ANILINE	239.3	6400	6.8E-03	1.8E-01
2-METHYLPHENOL	250.9	12000	7.2E-03	3.4E-01
4-METHYPHENOL	2043.0	120000	5.8E-02	3.4E+00
2,4-DIMETHYLPHENOL	5623.0	37000	1.6E-01	1.1E+00
NAPHTHALENE	351.2	1900	1.0E-02	5.4E-02
2-METHYLNAPHTHALENE	14.0	160	4.0E-04	4.6E-03
ACENAPHTHENE	4.6	43	1.3E-04	1.2E-03
DIBENZOFURAN	1.5	11	4.3E-05	3.1E-04
FLUORENE	1.4	9	4.0E-05	2.6E-04
PHENANTHRENE	1.5	10	4.3E-05	2.9E-04
Total PAH		2133		6.1E-02
Total LMW PAH		2133		6.1E-02
Total HMW PAH		0		0
Total Phenolics		206400		5.9E+00

potential for ultimate release can not be calculated with any degree of certainty. However, it is recognized that the potential exists and that (especially with regard to leaching of creosote) it could be important in the future.

Total PAH compounds were present at levels of up to 298 ng/l in the municipal supply. Eighteen of the 19 samples collected from the municipal supply contained low molecular weight PAH ranging from 9.1 to 298 ng/l. Elevated levels of these PAH compounds reflected a mix of compounds but were predominated by naphthalene, fluorene, phenanthrene and fluoranthene. The geometric mean concentration for the low molecular weight compounds was 75.8 ng/l. The high molecular weight PAH compounds were detected in seven of the 19 municipal supply samples and ranged up to 9 ng/l. The high molecular weight compounds detected in each of these seven samples were benzo(a)anthracene and chrysene with benzo(a)pyrene and benzofluoranthenes also detected in one sample. The geometric mean concentration of the high molecular weight PAH was approximately 1.2 ng/l.

Table 11-2 contains the levels of PAH compounds and associated exposures will be used for estimating the risks associated with drinking water from the municipal supply.

11.2 Exposure of Residents to Contaminants in Air

Section 7 of the Somers RI/FS presented data on measured air quality concentrations during plant operations and predicted air quality emissions and ambient concentration from volatilization of constituents in the CERCLA lagoon soils. Naphthalene was found to be the parameter with the greatest potential for impacting air quality downwind of the CERCLA lagoon. These data will be incorporated into the risk assessment. For exposure estimates, an average individual inhales approximately 20 m^3 of air during the course of a 24-hr day.

11.3 Exposure From Ingestion of Fish

Tissue analysis of two fish species (Dolly Varden and lake

TABLE 11-2

ESTIMATES OF EXPOSURES FROM DRINKING THE SOMERS MUNICIPAL SUPPLY WATER

	Somers Municipal System			
	Conc. (ng/l)		Exposure (mg/kg/day)	
	G. Mean	Max.	G. Mean	Max.
Naphthalene	15.0	190	4.3E-07	5.4E-06
Acenaphthalene	1.1	2.9	3.1E-08	8.3E-08
Acenaphthene	1.1	3	3.1E-08	8.6E-08
Fluorene	12.8	56	3.7E-07	1.6E-06
Phenanthrene	25.4	72	7.3E-07	2.1E-06
Anthracene	2.4	54	6.9E-08	1.5E-06
Fluoranthene	6.2	24	1.8E-07	6.9E-07
Pyrene	1.1	4	3.1E-08	1.1E-07
Benzo(a)Anthracene	1.2	5	3.4E-08	1.4E-07
Chrysene	1.3	7.4	3.7E-08	2.1E-07
Benzofluoranthenes	1.0	1.4	2.9E-08	4.0E-08
Benzo(a)pyrene	1.1	3	3.1E-08	8.6E-08
Total PAH	77.5	298	2.2E-06	8.5E-06
Low Molecular Wght PAH	75.8	298	2.2E-06	8.5E-06
High Molecular Wgt PAH	1.6	9	4.6E-08	2.6E-07

G. Mean = Geometric Mean

whitefish) were carried out on samples taken from Kalispell Bay, near the tie plant. Results are presented and discussed in Section 9.1. Metal concentrations were found to be within natural levels and concentrations of PAH above instrument detection limits (0.5 mg/kg) were not found in any of the fish tissues.

As was the case with drinking water considerations, there is the potential that increased quantities of PAH compounds may be released to the lake in the future via runoff, discharge of contaminated groundwater from around the swamp pond, and possible leaching of non-aqueous phase liquids (creosote) from the swamp/-beach area. The magnitude of these sources or potential for ultimate release can not be calculated with certainty. However, it is recognized that the potential exists and that (especially with regard to leaching of creosote) it could be important in the future.

Measurements of PAH in fish have revealed that levels are below 0.5 mg/kg. These low levels are consistent with the low levels that have been measured in surface sediment samples in the lake (less than 1.5 mg/kg) and in the water (generally on the order of a few ng/l).

PAH compounds are typically bioaccumulated in fish tissues to levels in excess of those that exist in the water column but generally less than those that occur in sediments. The degree to which a fish will bioaccumulate PAH compounds depends on the lipid (fat) content of the fish and the lipophilic nature of the particular PAH compound. In most cases, accumulation from water is more efficient than from food or sediment. Bioavailability of sedimentary PAH is critical in determining exposure for animals, especially benthic and demersal species. Bioavailability is strongly influenced by association of PAH with colloidal or particulate material. Sorption-desorption equilibria of PAH with particulate matter clearly indicate that some fraction of PAH is in solution in sediment pore water. Therefore, animals compete with sedimentary material for the available PAH in solution. The dynamics of these competing processes determines, in part, the

rate of PAH uptake. Most animals are able to degrade PAH to more polar metabolites and excrete them (Neff 1979). Thus, food-chain biomagnification of PAH occurs only to a very limited extent, if at all.

The data base on bioconcentration of high molecular weight PAH compounds from water is sparse. An estimate of potential accumulation can be obtained using the log K_{ow} model based on the octanol-water partition coefficient (K_{ow}). This coefficient is a measure of hydrophobicity of a chemical but ignores other properties that might affect bioaccumulation of a substance. MacKay (1982) has proposed a simple relationship between surface water concentrations and fish tissue levels as follows:

$$C_f/C_w = 0.048\ K_{ow}$$

where C_f = concentration of contaminant in the fish
C_w = concentration of contaminant in surface water
K_{ow} = octanol/water partition coefficient

Using this equation, body burdens in fish tissues can be estimated. The maximum concentrations in ng/l (ppt) observed in Flathead Lake during the April 1987 sampling have been used to estimate tissue concentrations in mg/kg (ppm) and exposures in mg/kg/day as shown below. For exposure analysis, it was assumed that 6.5 gms of fish/day would be consumed by an adult with a body weight of 70 kg.

	Lake (ng/l)	Kow	Fish (mg/kg)	Exposure (mg/kg/day)
Naphthalene	47	3890.5	0.0088	8.2x10-4
Acenaphthalene	3.9	12022.6	0.0022	2.1x10-4
Phenanthrene	6.1	25118.9	0.0074	6.8x10-4
Fluoranthene	10	316227.8	0.152	1.4x10-2

The above the fish tissue estimates are significantly lower than the detection limit of PAH compounds in tissues of fish (0.5 mg/kg). The estimates are considered reflective of typical ambient conditions that may occur under general exposure to PAH

compounds. With this qualification in mind, the estimates will be used to examine risks posed by ingestion of fish from Flathead Lake.

11.4 Exposure by Direct Contact with Soils/Sediments

Soils and sediments at the site present a potential exposure route for direct contact with contaminated soils/sediment which are present at the ground surface. Because of the site's proximity to residential areas and despite its limited accessibility, it is possible that people (especially children) might periodically trespass and have direct contact with contaminated soils at the surface. Subsurface soils do not present an exposure potential unless and until those soils are exposed. This could occur if the site were developed and subsurface structures (e.g. basements, utilities, etc) were installed. Table 11-3 presents the maximum and geometric mean concentrations of surface and subsurface soils at the Somers site.

To evaluate the exposure under current conditions when children might trespass on the site, it was assumed that children would visit the site once a week from the ages of 5 to 12. A "worst case" residential use scenario was also considered where exposure to contaminated soils would occur daily over a lifetime of 70 years. The average ambient temperature in the Somers area exceeds 35 °F only seven months of the year. Frozen ground conditions and snow cover will restrict direct contact with soils. Therefore, each case above is assumed to occur for seven months/year. This is a conservative but reasonable assumption.

Along with frequency and duration of exposure to contaminated soils, an estimate of the quantity of soil associated with each exposure is required. Several investigators have published data on soil ingestion rates. Kimbrough et al (1984) provided one of the first estimates and is often cited. More recent work has shown that these initial estimates were probably overestimates. LaGoy (1987) presented a set of age-dependent soil ingestion rates as follows:

Table 11-3

SUMMARY OF SURFACE AND SUBSURFACE SOIL CONCENTRATIONS

Parameter, mg/kg	Surface Soils (1) Maximum	Surface Soils (1) Geometric Mean	Sub-surface Soils (2) Maximum	Sub-surface Soils (2) Geometric Mean
Naphthalene	500000	6.2	940	44.9
2-Methylnaphthalene	9400	4.8	440	21.5
Acenaphthylene	0.54	0.7	0.057	0.8
Acenaphthene	6600	4.1	240	21.8
Dibenzofuran	2700	4.0	160	11.4
Fluorene	2700	3.5	170	16.5
Phenanthrene	8000	4.1	300	25.5
Anthracene	3000	2.4	240	7.4
Fluoranthene	4700	14.1	640	14.6
Pyrene	33000	17.9	100	9.8
Chrysene	35	1.7	21	1.8
Benzo(a)anthracene	30	1.4	52	2.4
Benzo(b)fluoranthene	22	1.7	12	1.3
Benzo(k)fluoranthene	0.13	0.7	20	1.3
Benzo(a)pyrene	45	1.7	14	1.2
Indeno(1,2,3-cd)pyrene	1.9	1.2	ND	ND
Dibenzo(a,h)anthracene	0.89	0.9	ND	ND
Benzo(g,h,i)perylene	2.3	1.3	ND	ND

---

(1) Based on results of HSL CERCLA Lagoon sample, CTP-1, GTP-1, CTP-8, and maximum slough concentrations

(2) Based on results of

BH-3 15-16.5′	CTP-1 3-5′	CTP-1 11-12′	CTP-2 6-8′	
CTP-3 9-12′	CTP-4 8-9′	CTP-5 7-9′	CTP-5 8-9′	CTP-6 8-10′
	CTP-7 9-10′	CTP-8 8-9′	S-88-2 11-13′	S-88-2 11-13′

ND - Parameter not reported present above detection limits

Age (years)	Avg. Weight (kg)	Ingestion (mg/day)
0-1	10	50
1-6	15	100
6-11	30	50
over 11	70	50

Hawley (1985) estimated that children ingest approximately 50 mg/day in a typical play visit. Pauley (1987) suggests that an ingestion rate of 1 to 10 mg/day for adolescents and adults is a reasonable and supportable value. Based on these studies, the following values have been incorporated into the Somers study:

Age (years)	Avg. Weight (kg)	Ingestion (mg/day)
0-1	10	50
1-6	15	100
6-11	30	50
11-18	70	50
over 18	70	10

Another important consideration in assessing exposures from soils is the matrix effect. Matrix effects refer to the difference in the bio-availability of a compound from soils as compared to the bio-availability of the chemical as administered in the animal assays on which effects are based. It is clear that the matrix in which PAH are administered affects bio-availability. Although no data were found on PAH in soils, data are available on the availability of tetrachlorodibenzodioxin (TCDD) in soil. Umbreit et al (1986) report on the bio-availability of dioxin from a 2,4,5-T manufacturing site. They reported bio-availabilities of less than 5 percent to 21.3 percent and cite differences in organic soil content and in the mode of deposition as affecting the availabilities. Poiger and Schlatter (1980) investigated the absorption of TCDD administered in a 50% ethanol solution, in soil slurries aged for 10-15 hours and for 8 days, and in an aqueous suspension of activated carbon. The availability ranged from 36.7% in ethanol to less than 0.07 % for activated carbon. The aged soil slurries were found to provide an available frac-

tion of 24.1 percent at 10-15 hrs to 16 percent at 8 days. The authors suggest that the binding of the TCDD to soil particles increased with time and hence decreased the availability. A matrix effect value of 20 percent is therefore believed reasonable for the Somers site.

Exposure assessments may also consider biodegradation effects, recognizing that the soil concentrations will not remain constant over a 70 year period of exposure. Extensive data exist on the degradation of PAH under induced optimal conditions (e.g. high oxygen, high temperatures, nutrient addition, etc). These data indicate that the lower molecular rate PAH degrade more quickly than the higher molecular weight compounds: e.g. naphthalene has a half life of about three months while benzo(a)pyrene has a half life approaching 1 year. Estimates of natural degradation rates show a similar variation among compounds and also among media. To provide a worst case assessment, no degradation will be assumed in this effort.

Table 11-4 presents the exposures in (mg/kg/day) calculated for the exposure pathway of direct contact with and ingestion of soils. The average daily lifetime exposure was determined using an electronic spreadsheet which calculated age-dependent exposures as described above then averaging them over the exposure period (70 years for residential and 7 years for current use).

11.5 Exposure by Direct Contact While Swimming

Despite the restricted access to Somers Beach, some people may use trespass and use this area for recreational purposes. The exposure routes of potential concern considered here are direct contact with the water and sediments skin while swimming, playing, etc. on the beach.

During the time period that swimming and most recreational activities would occur (summer months), the lake is at its highest level and water covers the beach sediments near the former swamp pond. Since there is exposure to potentially contaminated sediments during the summer months, direct contact is not a route

TABLE 11-4

ESTIMATED EXPOSURES FROM INGESTION OF SOILS

Average Lifetime Exposure (mg/kg/day)

	Surface Soils				Subsurface Soils	
	Residential		Current Use		Residential	
	Maximum	G. Mean	Maximum	G. Mean	Maximum	G. Mean
Naphthalene	5.1E-02	6.3E-07	1.8E-02	2.2E-07	9.6E-05	4.6E-06
2-Methylnaphthalene	9.6E-04	4.9E-07	3.4E-04	1.7E-07	4.5E-05	2.2E-06
Acenaphthylene	5.5E-08	7.1E-08	2.0E-07	2.5E-08	5.8E-09	8.1E-08
Acenaphthene	6.7E-03	4.2E-07	2.4E-04	1.5E-07	2.4E-05	2.2E-06
Dibenzofuran	2.8E-04	4.1E-07	9.8E-05	1.4E-07	1.6E-05	1.2E-06
Fluorene	2.8E-04	3.6E-07	9.8E-05	1.3E-07	1.7E-05	1.7E-06
Phenanthrene	8.1E-03	4.2E-07	2.9E-04	1.5E-07	3.1E-05	2.6E-06
Anthracene	3.0E-04	2.4E-07	1.1E-04	8.7E-08	2.4E-05	7.5E-07
Fluoranthene	4.8E-04	1.4E-06	1.7E-04	5.1E-07	6.5E-05	1.5E-06
Pyrene	3.4E-03	1.8E-06	1.2E-03	6.5E-07	1.0E-05	1.0E-06
Chrysene	3.6E-06	1.7E-07	1.3E-06	6.2E-08	2.1E-06	1.8E-07
Benzo(a)anthracene	3.0E-06	1.4E-07	1.1E-06	5.1E-08	5.3E-06	2.4E-07
Benzo(b)fluoranthene	2.2E-06	1.7E-07	8.0E-07	6.2E-08	1.2E-06	1.3E-07
Benzo(k)fluoranthene	1.3E-08	7.1E-08	4.7E-09	2.5E-08	2.0E-06	1.3E-07
Benzo(a)pyrene	4.6E-06	1.7E-07	1.6E-06	6.2E-08	1.4E-06	1.2E-07
Indeno(123-cd)pyrene	1.9E-07	1.2E-07	6.9E-08	4.4E-08	0.0E+00	0.0E+00
Dibenzo(ah)anthracene	9.1E-08	9.2E-08	3.2E-08	3.3E-08	0.0E+00	0.0E+00
Benzo(g,h,i)perylene	2.3E-07	1.3E-07	8.3E-08	4.7E-08	0.0E+00	0.0E+00

of exposure.

Ingestion of water during swimming could occur but would be much less than the 2 liters/day assumed in the drinking water exposure assessment. Therefore, the drinking water scenario presents a worst case assessment of this exposure. This is therefore not determined to be a significant exposure pathway and will not be considered further.

11.6 Public Heath Risk Assessment

This section provides quantitative estimates, where possible, concerning the potential human health risks associated with the Somers Tie Treating Plant.

For compounds that are handled as non-carcinogens, the exposure levels are compared to acceptable daily intake values for acute and chronic exposures in accordance with the EPA suggested methodology. For compounds that are handled as carcinogens, risks are estimated from estimated levels and duration of exposure and by estimates of carcinogenic potencies. The EPA Cancer Assessment Group is used as a source of potency information.

The EPA has published no acceptable intake values for noncarcinogenic PAH compounds. A value has been published for phenol, a compound which was found to be present in the groundwater wells downgradient of the CERCLA Lagoon. Using the same methodology and approach as EPA, Environ (1986) estimated acceptable intake values for select PAH. A summary of these values and the underlying bases for the numbers is presented in Table 11-5. Very large safety factors (1,000 to 10,000) were used to estimate these values. Because of the margin of safety built into these values, exceedance of the number has no immediate meaning with respect to specific health effects or the frequency or magnitude of the health effects. However, exceedance of the number should serve as an indicator that the potential for unacceptable exposure does exist and measures should be taken to limit that exposure.

TABLE 11-5

ESTIMATED AIC VALUES FOR SELECTED PAH COMPOUNDS USING RFD APPROACH

Chemical	Estimated AIC Value (mg/kg/day)	Basis for Number	Target Organ(s)	Observed Effect	Uncertainty Factor
Acenaphthene	0.2	LOAEL for rats; oral dose of 1.0 g/kg/day and higher doses of 2.0 g/kg/day (Knobloch et al., 1969)	Liver, kidney, bronchi	Gain in liver weight; increase in enzyme activity, increased urine flow; morphological damage to kidney and liver and mild bronchitis at higher dose	5,000 includes 1,000 to account for intra- and interspecies variability and a factor of 5 to compensate for use of LOAEL rather than NOAEL
Naphthalene	0.053	Subchronic NOAEL for mice of 53 mg/kg/day (Shopp et al., 1984) administered by gavage	Spleen lung, thymus, body weight	Decreased spleen and thymus weight; increased lung weight	1,000 accounts for intra- and interspecies variability
Naphthalene	0.0053	NOAEL for mice of 5.3 mg/kg/day (Shopp et al, 1984) administered by gavage	Serum protein levels	Increase in serum protein levels	1,000 accounts for intra- and interspecies variability
Acenaphthalene	0.03	Subchronic LOAEL for rats of 300 mg/kg/day as an oral dose	Body weight, liver, kidney	Decrease in organ weight; minor degeneration; increased enzyme activity; increased urine flow; study considered inadequate	10,000, 1,000 accounts for intra and interspecies differences; additional 10 accounts for use of LOAEL and method problems
Pyrene	0.06	Subchronic LOAEL based on oral dose of 120 mg/kg/day (White and White, 1939)	Liver	Slightly fatty and enlarged liver	2,000, 1,000 accounts intra- and interspecies differences; additional 2 accounts for use of LOAEL

EPA has published a potency factor for only one of the known or suspected carcinogenic PAH, benzo(a)pyrene. This value has since been withdrawn by the agency because of problems with the method used to derive the number. EPA is currently reviewing a new report (Clements 1987b) in which a revised potency factor for benzo(a)pyrene has been derived. Although this value is still considered draft and is under EPA review, it is expected to eventually replace the original EPA estimate.

In estimating the risks associated with other carcinogenic PAH compounds, the general practice is to utilize B(a)P as a surrogate for the other compounds. It is well recognized that this will overestimate the risks because B(a)P is one of the most potent chemicals among PAHs. Several groups (Chu and Chen 1984; Clements 1987a,b) have been working toward developing relative potency factors for other PAH.

The available data on acceptable intake values and carcinogenic potency estimates for the parameters identified in the exposure assessment are summarized in Table 11-6.

11.6.1 Risks Associated with Drinking Water

The exposure analysis calculated intakes from the existing private and municipal water supplies as well as from a hypothetical new water supply well installed immediately downgradient from the CERCLA lagoon. Estimates of health risks associated with those exposures is now presented.

11.6.1.1 Private Wells

As presented in the exposure assessment, naphthalene, acenaphthalene, fluorene and benzofluoranthenes were detected in private water supply samples. Naphthalene, acenaphthalene, and fluorene are noncarcinogenic PAH and are therefore evaluated by comparison of exposure values to AIC values. The results are shown below for the maximum exposure values detected in private supplies.

TABLE 11-6

SUMMARY OF ACCEPTABLE INTAKE VALUES AND CARCINOGENIC POTENCY FACTORS

Parameter	Acceptable Daily Intake (mg/kg/day) Oral	Carcin. Potency Factor (mg/kg/day)-1 Oral	Carcin. Potency Factor (mg/kg/day)-1 Inhalation	Relative Potency (Clements)
Naphthalene	0.05 to 0.005			
Acenaphthalene	0.03			
Acenaphthene	0.20			
Fluorene	0.07 *			
Phenanthrene	0.07 *			
Anthracene	0.07 *			
Fluoranthene	0.07 *			
Pyrene	0.06			
Benzo(a)Anthracene		11.5	6.11	0.1450
Chrysene		11.5	6.11	0.0044
Benzo(b)fluoranthene		11.5	6.11	0.1400
Benzo(k)fluoranthene		11.5	6.11	0.0660
Benzo(a)pyrene (EPA)		11.5	6.11	
Benzo(a)pyrene (Clements)		3.22	0.4533	1.0000
Indeno(123-cd)pyrene		11.5	6.11	0.2320
Dibenzo(ah)anthracene		11.5	6.11	1.1100
Benzo(ghi)perylene		11.5	6.11	0.0100
Phenol	0.04			
Zinc	0.21			

---

* Value assumed equal to average AIC for other noncarcinogenic PAH

Clements (1987 a,b)

Parameter,mg/kg/day	Maximum	AIC
Naphthalene	1.1x10-6	5.3x10-3
Acenaphthalene	2.2x10-7	3.0x10-2
Fluorene	5.7x10-8	7.0x10-2

As can be seen, no chronic health effects are predicted from this level of exposure. Benzofluoranthenes were calculated to present a maximum exposure of $1.0x10^{-7}$ mg/kg/day. Using the benzo(a)-pyrene potency factor of 11.5 mg/kg/day^{-1}, an risk level of $1.1x10^{-6}$ is calculated. This means there could be one excess cancer death in a population of a million people who drank two liters of this water every day for a lifetime of 70 years. Using the draft revised benzo(a)pyrene potency factor of 3.22 mg/kg/day^{-1}, the calculated risk drops to 3 excess cancer deaths in 10 million people. If one also considers that benzofluoran-thenes are less potent that benzo(a)pyrene by factors of 0.066 to 0.14, the estimated risk decreases even more. Given these factors and the use of the maximum detected concentration, the consumption of water from private wells in Somers is not consid-ered to present a significant risk under current conditions.

11.6.1.2 Municipal Supply

Table 11-7 presents the calculated exposures to noncarcin-ogenic and carcinogenic PAH detected in the municipal water supply. As before, all noncarcinogenic exposures are well below the acceptable intake values. Carcinogenic risks range from 3 in a million (3x10-6) for the maximum reported total carcinogenic PAH concentration to 5 in 10 million for the geometric mean concentration of total carcinogenic PAH. Use of the revised draft benzo(a)pyrene potency factor would reduce the maximum risk to 8 in 10 million. Consideration of relative potencies for benzo(a)anthracene, chrysene and benzofluoranthenes would further reduce the calculated risk levels.

TABLE 11-7

ASSESSMENT OF EXPOSURE RISK FROM MUNICIPAL SUPPLY WATER

	Somers Municipal System					
	Conc. (ng/l)		Exposure (mg/kg/day)		Acceptable Intake	
	G. Mean	Max.	G. Mean	Max.	(mg/kg/day)	
Naphthalene	15.0	190	4.3E-07	5.4E-06	5.3E-03/5.3E-02	
Acenaphthalene	1.1	2.9	3.1E-08	8.3E-08	3.0E-02	
Acenaphthene	1.1	3	3.1E-08	8.6E-08	2.0E-01	
Fluorene	12.8	56	3.7E-07	1.6E-06	7.0E-02	
Phenanthrene	25.4	72	7.3E-07	2.1E-06	7.0E-02	
Anthracene	2.4	54	6.9E-08	1.5E-06	7.0E-02	
Fluoranthene	6.2	24	1.8E-07	6.9E-07	7.0E-02	
Pyrene	1.1	4	3.1E-08	1.1E-07	6.0E-02	
					Max. Risk Level	
Benzo(a)Anthracene	1.2	5	3.4E-08	1.4E-07	1.6E-06	
Chrysene	1.3	7.4	3.7E-08	2.1E-07	2.4E-06	
Benzofluoranthenes	1.0	1.4	2.9E-08	4.0E-08	4.6E-07	
Benzo(a)pyrene	1.1	3	3.1E-08	8.6E-08	9.9E-07	
Total High Molecular Weight PAH	1.6	9	4.6E-08	2.6E-07	3.0E-06	3.0E-06

G. Mean = Geometric Mean

11.6.1.3 Hypothetical New Well

Exposure to organic compounds present in the groundwater downgradient of the CERCLA lagoon was estimated under the assumption that someone could install a new water supply well there. No carcinogenic PAH were detected in the groundwater samples from Wells S-88-1, 88-2 and 88-3. Noncarcinogenic PAH and phenolic compounds were present however. Table 11-8 provides a comparison of the maximum and geometric mean exposures to acceptable daily intakes. Maximum exposures would exceed "acceptable" levels for phenol and naphthalene. Therefore, the installation of a new water supply well in the vicinity of these groundwater monitoring wells cannot be recommended.

11.6.2 Risks Associated with Exposure to PAH Compounds in Air

Chapter 7 presented predicted concentrations of naphthalene and other non-carcinogenic PAH in ambient air due to volatilization from the CERCLA lagoon. Noncarcinogenic compounds are evaluated with reference to acceptable daily intake levels. As shown in Tables 11-5 and 11-6, the acceptable intake for naphthalene is from 0.0053 to 0.053 mg/kg/day. For a 70 kg individual, this would correspond to an acceptable intake of 0.37 to 3.7 mg/day of naphthalene. An average individual inhales approximately 20 m^3 of air during the course of a 24-hr day. Thus, an "acceptable" level of naphthalene in air is 18 to 180 ug/m^3. Predicted air concentrations of naphthalene ranged from 25 to 175 ug/m^3 at a distance of 100 meters down wind of the CERCLA Lagoon. Therefore, there may be some concern for systemic (noncarcinogenic) risks due to elevated levels of naphthalene in air. Because the acceptable intake level was arrived at by applying a large safety factor (1000) to observations of effects of naphthalene on rats, it is unclear what effects, if any, these levels would have on humans. Nevertheless, the results suggest that actions taken to minimize the presence of naphthalene in surface soils and thus minimize the potential for off-site migration would help ensure that effects associated with this chemical are minimized. None

TABLE 11-8

ASSESSMENT OF EXPOSURE RISK FROM GROUNDWATER

	Geometric Mean (ug/l)	Maximum (ug/l)	Exposure (mg/kg/day) G. Mean	Exposure (mg/kg/day) Maximum	Acceptable Intake (mg/kg/day)
PHENOL	271.2	31000	7.7E-03	8.9E-01	4.0E-02
ANILINE	239.3	6400	6.8E-03	1.8E-01	ND
2-METHYLPHENOL	250.9	12000	7.2E-03	3.4E-01	ND
4-METHYPHENOL	2043.0	120000	5.8E-02	3.4E+00	ND
2,4-DIMETHYLPHENOL	5623.0	37000	1.6E-01	1.1E+00	ND
NAPHTHALENE	351.2	1900	1.0E-02	5.4E-02	5.3E-03/5.3E-02
2-METHYLNAPHTHALENE	14.0	160	4.0E-04	4.6E-03	ND
ACENAPHTHENE	4.6	43	1.3E-04	1.2E-03	2.0E-01
DIBENZOFURAN	1.5	11	4.3E-05	3.1E-04	ND
FLUORENE	1.4	9	4.0E-05	2.6E-04	7.0E-02
PHENANTHRENE	1.5	10	4.3E-05	2.9E-04	7.0E-02

of the other PAH compounds modelled would pose a potential health risk to residents.

11.6.3 Risks Associated with Exposure to Soils/Sediments

Estimates of health risks associated with the direct contact with soils/sediments at the site were obtained for noncarcinogenic and carcinogenic compounds. Table 11-9 compares the estimated exposure and acceptable daily intake for noncarcinogenic compounds and presents the estimated risk levels for carcinogenic compounds.

For noncarcinogenic compounds, naphthalene exposures to maximum surface soil concentrations (i.e. CERCLA lagoon soils) under both the current use and hypothetical residential use scenarios are greater than the lower acceptable intake. The calculated lifetime average daily exposure to naphthalene under residential use is 0.05 mg/kg daily while the average 7 year exposure is 0.02 mg/kg/day. As was shown in tables 11-5 and 11-6, acceptable intakes of naphthalene are 0.053 to 0.005 mg/kg/day. No other noncarcinogenic compounds exceed acceptable intakes.

Maximum risks to benzo(a)pyrene in surface soils are 5 in 100,000 under the residential scenario and 2 in 100,000 under the current use scenario. If one assumes all carcinogenic PAH are as potent as benzo(a)pyrene, the calculated risks of death from cancer due to ingestion of CERCLA lagoon soils increases to 2 in 10,000 under the residential scenario and 6 in 100,000 under the current use scenario. Using the geometric mean surface soil concentration and exposures, the risks from ingestion of benzo-(a)pyrene under presumed residential and current use are 2 in 1 million and 7 in 10 million, respectively. Again assuming all carcinogenic PAH are as potent as benzo(a)pyrene, the risks increase to 2 in 100,000 and 5 in one million for residential and current use situations.

TABLE 11-9

ESTIMATE OF EXPOSURE RISK FROM SOIL INGESTION

Average Lifetime Exposure (mg/kg/day)

	Surface Soils				Subsurface Soils		
	Residential		Current Use		Residential		Acceptable Intake
	Maximum	G. Mean	Maximum	G. Mean	Maximum	G. Mean	(mg/kg/day)
Naphthalene	5.1E-02	6.3E-07	1.8E-02	2.2E-07	9.6E-05	4.6E-06	5.3E-02/5.3E-03
2-Methylnaphthalene	9.6E-04	4.9E-07	3.4E-04	1.7E-07	4.5E-05	2.2E-06	ND
Acenaphthylene	5.5E-08	7.1E-08	2.0E-07	2.5E-08	5.8E-09	8.1E-08	3.0E-02
Acenaphthene	6.7E-03	4.2E-07	2.4E-04	1.5E-07	2.4E-05	2.2E-06	2.0E-01
Dibenzofuran	2.8E-04	4.1E-07	9.8E-05	1.4E-07	1.6E-05	1.2E-06	ND
Fluorene	2.8E-04	3.6E-07	9.8E-05	1.3E-07	1.7E-05	1.7E-06	7.0E-02
Phenanthrene	8.1E-03	4.2E-07	2.9E-04	1.5E-07	3.1E-05	2.6E-06	7.0E-02
Anthracene	3.0E-04	2.4E-07	1.1E-04	8.7E-08	2.4E-05	7.5E-07	7.0E-02
Fluoranthene	4.8E-04	1.4E-06	1.7E-04	5.1E-07	6.5E-05	1.5E-06	7.0E-02
Pyrene	3.4E-03	1.8E-06	1.2E-03	6.5E-07	1.0E-05	1.0E-06	6.0E-02
	Risk Level Using CPF = 11.5 (mg/kg/day)-1						
Chrysene	4.1E-05	2.0E-06	1.5E-05	7.1E-07	2.4E-05	2.1E-06	
Benzo(a)anthracene	3.5E-05	1.6E-06	1.3E-05	5.9E-07	6.1E-05	2.8E-06	
Benzo(b)fluoranthene	2.5E-05	2.0E-06	9.2E-06	7.1E-07	1.4E-05	1.5E-06	
Benzo(k)fluoranthene	1.5E-07	8.2E-07	5.4E-08	2.9E-07	2.3E-05	1.5E-06	
Benzo(a)pyrene	5.3E-05	2.0E-06	1.8E-05	7.1E-07	1.6E-05	1.4E-06	
Indeno(123-cd)pyrene	2.2E-06	1.4E-06	7.9E-07	5.1E-07	0.0E+00	0.0E+00	
Dibenzo(ah)anthracene	1.0E-06	1.1E-06	3.7E-07	3.8E-07	0.0E+00	0.0E+00	
Benzo(g,h,i)perylene	2.6E-06	1.5E-06	9.5E-07	5.4E-07	0.0E+00	0.0E+00	
Total	1.6E-04	1.2E-05	5.7E-05	4.4E-06	1.4E-04	9.2E-06	

11.6.4 Risks Related to Ingestion of Fish

As discussed in the exposure analysis, levels of metals in the water are not inconsidered to pose any human health risks either as a result of direct ingestion of water or via ingestion of fish.

Estimates of PAH compounds in fish were made from measurements in water. Estimated levels are judged to be comparable to those in ambient freshwater bodies not receiving heavy contamination by PAHs. Therefore, this qualification should be kept in mind during the following discussion.

These risks were evaluated by comparing the estimated intake of PAH compounds via ingestion of fish caught in Flathead Lake with the acceptable intake values estimated for the compounds. For this analysis it was assumed that the ingestion rate of fish was 6.5 grams/day and that the body weight of an average individual was 70 kg.

	Exposure (mg/kg/day)	Acceptable Intake (mg/kg/day)
Naphthalene	8.2x10-4	5.3x10-3 to 5.3x10-2
Acenaphthalene	2.1x10-4	3.0x10-2
Phenanthrene	6.8x10-4	7.0x10-2
Fluoranthene	1.4x10-2	7.0x10-2

As is clear from the above, levels of these PAH compounds in fish are well within acceptable levels under existing conditions.

11.7 Summary of Risks

Risks have been characterized for major exposure routes. Below we discuss these risks in terms of existing conditions as well as potential for future exposure.

Drinking Water - At present, there is little existing risk associated with contaminants in drinking water from private and municipal water supplies. However, it is recognized that there is a potential for introduction of PAH compounds to Flathead Lake via the drainage ditch and the swamp pond areas at the site. The drainage ditch itself is contaminated and could also serve as a

conduit for contaminants from the CERCLA lagoon. Development of a new groundwater supply within the immediate vicinity (a few hundred feet) of the CERCLA lagoon could present health risks from noncarcinogenic PAH. Although no carcinogenic PAH were detected in the groundwater monitoring wells in this area, it is possible that they might be present at levels below the detection limit and could also pose adverse health risks to the consumers of this hypothetical well. Thus, to the extent that existing areas of soil and groundwater contamination are addressed, the potential for contamination of groundwater and of potential future water supplies would be further minimized.

Air - An analysis was conducted which indicated that elevated levels of PAH compounds (primarily naphthalene) could exist off-site due to volatilization of contaminants from the CERCLA lagoon soil. The estimated levels of naphthalene would exceed one measure of acceptable daily intakes and could therefore present a health concern. Emissions from the site have been greatly reduced since the tie treating operations have been terminated. The estimates presented in this report are considered highly conservative with regard to present exposure levels in air. Thus, remediation is not contemplated to be necessary based on potential for exposure via the atmosphere. However, if remedial measures are contemplated for other reasons, it is suggested that an evaluation of these methods include an assessment of the impacts upon local air quality. Such an assessment should include a consideration of volatiles as well as fugitive dusts.

Soils - Risks associated with exposure of children playing at the site to contaminated surface soils is judged to be the most important exposure pathway based on an analysis of current risks. A hypothetical scenario was also evaluated in which the site was developed for residential use prior to any remedial actions. For noncarcinogenic compounds, naphthalene exposures to

maximum surface soil concentrations (i.e. CERCLA lagoon soils) under both the current use and hypothetical residential use scenarios are greater than the acceptable intake and pose a potential health concern.

Maximum carcinogenic risks from ingestion of surface soils assuming all carcinogenic PAH are as potent as benzo(a)pyrene, are 2 in 10,000 under the residential scenario and 6 in 100,000 under the current use scenario. Using the geometric mean surface soil concentration and exposures, the risks from ingestion of carcinogenic PAH (again using the overly conservative assumption that all carcinogenic PAH are as potent as benzo(a)pyrene), the risks of cancer deaths due to soil ingestion are 2 in 100,000 and 5 in one million for residential and current use situations.

Based on these data, and the ability of children to gain access to contaminated areas, it is recommended that the potential risk to human health posed by maximum surface soil concentrations (i.e., CEECLA lagoon soils) be reduced through remedial actions.

Ingestion of Fish - Risks associated with ingestion of fish were estimated from data on levels of PAHs in Flathead Lake. Measurements of the fish themselves indicated that levels were below detection (<0.5 mg/kg). Concentrations in fish estimated from water concentrations were also less than 0.5 mg/kg. At present, the exposure associated with ingestion of tissue from Flathead Lake fish is judged acceptable. There is, however, a potential for long-term input of PAH compounds to the lake via runoff through the ditch, possible seepage through the beach front, and contaminated groundwater from the swamp pond area. Thus, there is a potential for increased risks in the future.

12. ENVIRONMENTAL EXPOSURES

The assessment of environmental exposures considers the potential for uptake of contaminants through the food chain to aquatic life, ruminants and wildlife. This assessment is based on literature data reviews as well as the actual sampling and analysis of both aquatic life and waterfowl species.

12.1 Plant Uptake of PAH

PAH are widespread in soils as well as in crops, plants, and algae in the natural environment. Natural background levels of PAH are, in general, greatest in leafy materials of plants. Sims and Overcash (1983) reported that tree leaves contained 22 to 88 parts per billion (ppb) and cereal grains 48 to 66 ppb PAH. The source of PAH in plants is subject to much debate. Elevated benzo(a)pyrene (BaP) has been found in soils and in above-ground vegetation adjacent to urban areas (Kolar et al. 1975 and Smirnov 1970 as cited in Sims and Overcash 1983). However, other researchers have shown that levels of PAH are independent of area of origin (Hancock et al. 1970, Graf and Diehl 1976, and Borneff et al. 1968, as cited in Sims and Overcash, 1983).

According to Sims and Overcash (1983), the presence of PAH in plants is a result of airborne deposition, biochemical synthesis, and plant-uptake from soil. Much of the plant-uptake research has been conducted with BaP and is not necessarily representative of other PAH (Sims and Overcash 1983). In studies with BaP, the ratio of crop/soil (c/s) residues of BaP was higher in stems and straw when compared to the seed concentration. This indicated biomagnification of BaP but not as a direct function of soil concentration. While biomagnification of BaP and 3,4 benzofluoranthene is evident, other constituents of PAH may not be taken up by plants. It may be that biomagnification of PAH is not only compound specific but also plant specific. The fate and impacts of PAH in terrestrial plant systems have recently been reviewed by Santodonato et al. (1981). These reviews conclude

that the principal mechanism of PAH uptake by plants is by adsorption rather then absorption. For example, Harms (1975) showed that less the 0.2 percent of C14 BaP found in the roots of wheat was translocated to the shoots, and Blum and Swarbrich (1975) found no appreciable translocation of C14 BaP from the roots in green beans, cantaloupes, and cotton. In other words, airborne PAH will accumulate on the surface of roots and leaves, but in soil contaminated areas very little PAH will be translocated from the roots to the shoots. Thus, for crops grown in contaminated soil, PAH concentrations are likely to be higher in or on the roots than in above-ground plant parts. The reverse can be true, however, in areas with high levels of fallout of PAH-contaminated particulate matter.

Plants appear to demonstrate the ability to metabolize PAH. This metabolism or degradation of PAH may take place regardless of the route of entry into the plant (leaves or roots). Plants including alfalfa, rye, orchard grass, and vetch were found to metabolize BaP that was assimilated by these plants (Durmishidze et al. 1974 as cited by Sims and Overcash 1983).

In summary, it has been demonstrated that plants may incorporate some PAH into their tissues. However, biomagnification levels are quite low. Coupled with low biomagnification levels and demonstrated metabolism of certain constituents of PAH, the potential for hazard to food chain consumers seems minimal.

12.2 Cattle Uptake of PAH

At one time, a portion of the study area was used for the grazing of cattle. The area used for grazing included a portion of the old discharge ditch which lead to the concern that the cattle may have ingested PAH. Although no cattle currently graze in the area, this analysis was conducted to see what, if any, risks might have been associated with this activity.

12.2.1 Literature Review

While there is an extensive body of literature on the

effects of PAH on laboratory animals, there is little data on the impacts to ruminant animals. Animals are often exposed to creosote from treated lumber and posts. Toxicosis of PAH from creosote is usually a result of exposure from chewing treated lumber (Reynolds and Stedelin 1982). In many cases the reported poisonings resulted from exposures in which pentachlorophenol was used in conjunction with creosote.

In a reported case of acute toxicosis (Reynolds and Stedelin 1982), a herd of Hereford cattle was grazed adjacent to a railroad tie treatment (creosote) plant. Heavy rains washed creosote into the pasture area and the cattle that were exposed to this wash-out event experienced weight loss, unthriftiness, and one death. Subsequent analyses identified matching creosote peaks in the HPLC (High Performance Liquid Chromatography) analyses for rumen content extracts for a water sample from the pasture and for a coal-tar creosote sample from a local lumber yard. While this incident does indicate an occurrence of acute toxicosis, it was most likely the result of water contaminated with creosote and forage which had creosote adsorbed into the foliage. Direct uptake of creosote from the soil by the plants and subsequent uptake by the cattle was probably not the pathway of poisoning in this incident.

Bioaccumulation of PAH from water in vertebrate organisms is considered to be short-term, unlike the long-term bioaccumulation that has been demonstrated for the persistent chlorinated organics (Versar, Inc. 1979). Numerous studies show that despite a high lipid solubility, PAH show little tendency for bioaccumulation in the fatty tissues of animals or man (EPA 1980a). Metabolism and excretion of PAH in the urine and feces of laboratory animals has been documented (Versar, Inc. 1979) further indicating that bioaccumulation is limited. The above discussion seems to indicate, therefore, that although uptake of PAH by cattle from existing contaminated forage and soil is possible, long-term accumulation of PAH appears unlikely.

It is known that PAH are metabolized, mainly in the liver,

by mammals and humans. The metabolism takes place biochemically by the cytochrome P-450-dependent microsomal mixed-function oxidase (MFO) system. The MFO system can metogolize certain PAH and forms reactive epoxide metabolites which have been implicated as carcinogenic in experimental mammals (Sims and Grover 1974; Sims 1976; Lehr et al. 1978; and others).

In addition to the above pathway, a second pathway for formation of carcinogenic metabolites is the conversion of PAH epoxide metabolites by epoxide hydrase to vicinal glycols which may lead to carcinogenic bioactivation.

Although no acute or chronic toxicity to cattle from eating grass and soil contaminated with PAH is expected, the low levels ingested were evaluated as a carcinogenic risk. Cases of cancer in cattle from PAH uptake are unknown and no ingestion limits have been set for cattle. However, considering the very low ambient water quality criteria set for humans, a very low dose may be sufficient to cause cancer in cattle if they were to ingest the contaminated grasses and soil over a long period of time. However, because of the limited life expectancy of cattle, it appears highly unlikely that most cattle could be exposed for a period of time long enough to experience a carcinogenic effect.

12.2.1 Potential for Cattle Uptake of PAH at Somers

The Phase I report (ERT 1985) presented PAH analysis of swamp sludge and ditch soil as sampled by Montana Department of Health and Environmental Services in February 1984. These samples were taken to establish worst-case conditions. These data showed that the ditch soil sample had a total PAH concentration of less than 1000 ppm or less than 0.1 percent

Using the crop to soil (c/s) ratios of soil PAH concentration to crop PAH concentrations (Borneff et al. 1973 as cited by Sims and Overcash 1983), an evaluation of potential plant uptake of PAH from soil was made. Assuming the soil in the drainage ditch had a PAH concentration of 1000 ppm and the c/s ratio is

less than one, then the grasses along the ditch might potentially contain less than 1000 ppm PAH.

A cattle uptake evaluation was made based on the above assumed amounts of PAH on the grasses and in the soils. Since there are no previous chronic animal toxicity studies upon which to base the evaluation, several assumptions were made based on toxicity information from the literature. Using a daily chronic intoxication dose for cattle of 0.5 gm/kg body weight (Amstutz 1980), a 100-kg calf would need to consume more than 50 gm of PAH or 50,000 gm of grass containing 0.1 percent PAH to meet this daily chronic dose. A 100-kg calf typically consumes only about 5 kg (5,000 gm) of grass per day (Rolstein 1986). Thus, the typical calf would consume only 5 gm of PAH if its daily diet was totally from grasses growing along the ditch, an order of magnitude less than the reported chronic dose of 50 gm.

Additionally, it is known that cattle do ingest soil when foraging. Ingested soil may constitute up to 14 percent of dry matter ingested (Connor 1984). Soil PAH concentrations are about the same as the estimated grass concentration (less 0.1 percent), so an additional intake of 0.7 kg (700 gm) of soil to the calf's system would result in an additional PAH consumption of about 0.7 gm. The impact of PAH fallout from the plant onto the surrounding grasses was also evaluated. As discussed in Chapter 6, the highest measured levels were at the retort at 370 ug/m3 (ppm) (total coal tar pitch volatiles). If it is assumed that this concentration were evenly dispersed on nearby fields it would result in a concentration of 370 ppm of PAH on grass. At this additional concentration (0.370 ppm plus 1000 ppm = 1370 ppm) a cow could still not ingest a sufficient amount of grass or soil to experience a toxic effect. It therefore seems very unlikely that a calf or cow in the pasture near the tie plant would ingest enough PAH to meet the chronic toxic dose.

Given the evidence of metabolism and excretion of PAH and lack of demonstrated accumulation of PAH in fatty tissues of vertebrate organisms (EPA 1980a), it seems unlikely that PAH

accumulation to any significant concentration would have occurred in cattle pastured on this grazing area.

In summary, unreasonably large volumes of contaminated grass and soil would be required to obtain a chronically toxic dose in cattle grazing along the ditch. Futhermore, evidence from the literature suggests that PAH are not only metabolized and excreted by vertebrate organisms, but they also do not appear to accumulate to any great extent in fatty tissues.

Carcinogenesis in cattle grazing on contaminated grasses and soils near the tie plant appears unlikely due to the relatively short possible exposure times. Human consumption of any beef containing small residues of PAH from cattle that may have grazed in the pasture near the tie plant would be very sporadic and may only occur once in a particular person's lifetime.

12.3 Aquatic Life Uptake of PAH and Zinc

Sediments from the slough adjacent to the tie plant have been found to contain elevated levels of zinc and PAH. The levels of zinc in the slough water are also elevated. PAH have been detected in Flathead Lake and beach samples have be found to contain PAH. These factors led to assessment of the potential impact of the constituents to the fisheries resource.

12.3.1 Literature Review of PAH Uptake

PAH have been detected in tissues of many freshwater organisms. Neff (1979) states that this is not surprising since PAH are highly lipophilic (retained in fatty tissues). Removal of PAH from tissues may be passive or active. Active removal would involve metabolic transformation of PAH to water-soluble metabolites and subsequent excretion.

Lu et al. (1977, as cited by Santodonato et al. 1981) determined that freshwater snails bioaccumulated benzo(a)pyrene (BaP) by a bioconcentration factor (BCF) of 4,860 over water concentrations. The same author found that mosquito larvae accumulated BaP 37 times the water concentration after a 3-day exposure.

Filter-feeding organisms, such as mollusks (snails and clams) and daphnids, probably bioaccumulate PAH from water at high rates due to the retention of PAH-sorbed particulate materials on which they feed. Unlike many vertebrate groups, organisms like snails lack metabolic processes to detoxify and excrete PAH (Santodonato et al. 1981).

Freshwater algae rapidly accumulate PAH to high concentrations in water (Soto et al. 1975, Dobroski and Epifanio 1980 as cited by Neff 1985). It is thought that this uptake may be absorption of PAH onto cell surfaces (Herbes 1977 as cited by Neff 1985). Fish, unlike bivalve mollusks, can metabolize and excrete PAH. However, PAH equilibrium concentrations (body burdens) increase with exposure to higher molecular weight PAH compounds. PAH accumulate selectively in gallbladder, liver, brain, visceral fats and spleen tissues (Lee et al. 1972; Statham et al. 1976; DiMichele and Taylor 1978, as cited by Neff 1985).

Accumulation of PAH by aquatic organisms from contaminated food appears to be more variable and less efficient than PAH uptake from water (Neff 1985). Most of research in the area, however, has been conducted with marine (saltwater) species. In studies with hard shell clams some accumulation (BCF = 2.26) of BaP was demonstrated from ingestion of PAH-contaminated food (Dobroski and Epifanio 1980, as cited by Neff 1985). Marine polychaete worms did not assimilate significant amounts of methylnaphthalene from contaminated foods (Rossi 1977 as cited by Neff 1985).

Crustaceans (shrimp, crabs), however, have been shown to accumulate PAH more efficiently from food than from water (Corner et al. 1976; Harris et al. 1977, as cited by Neff 1985). Neff (1985) states that there appear to be large differences in the ability to absorb and assimilate PAH from food by different species. Where assimilation of dietary PAH was observed, metabolism and excretion of PAH were rapid; futhermore, the potential for food-web biomagnification of PAH seems limited. For such biomagnification from the food web to occur the material must be

readily absorbed from food, and once assimilated, relatively resistent to metabolism or excretion.

In regard to PAH accumulation from sediments little research has been conducted on this mechanism (Neff 1985). Since many benthic organisms ingest sediment and remove organic materials from it as a source of nutrition, PAH adsorbed sediments could potentially be mobilized into the aquatic food chain. In studies with marine organisms, bioconcentration factors for PAH were 0.12 for blood, 4.1 for the stomach wall of worms, and 0.2 or less for clams (Lyes 1979; Roeijadi et al. 1978, as cited by Neff 1985). Accumulation of PAH from sediment, when it occurs at all, may be attributed in large part to uptake of PAH desorbed from sediment particles into the interstitial water (Neff 1985). Generally, sediment-adsorbed PAH are not readily assimilated by benthic invertebrates, however, few species have been studied (Neff 1979). Futhermore, nothing is known of PAH uptake from sediments by attached aquatic plants (macrophytes).

12.3.2 Literature Review of Zinc Uptake

Zinc is bioaccumulated by all organisms. One noteworthy aspect of bioaccumulation is that it occurs even in the absence of abnormally high zinc concentrations since it is an essential nutrient (Versar 1979).

Bioconcentration factors are the ratio derived from the concentration of the element in the aquatic organism (in ppm of wet weight) divided by the concentration of the element in water (in ppm). Versar (1979) determined these bioconcentration factors for zinc in freshwater environments: plants 4,000; invertebrates 40,000; chironomid larvae 30,000; and fish 1,000.

Mollusks have been the subject of most investigations on zinc in marine animals. Oysters are able to concentrate certain trace metals to levels greater than 100,000 times the ambient concentration in seawater. The zinc content of most seafoods ranges from 3 to 30 ppm while oysters contain 100 to greater than 2000 ppm. Studies indicate that oysters loose zinc rapidly if

they are transplanted into water with low zinc content. A linear decrease from about 1000 ppm to about 100 ppm over a four month period was observed (National Research Council 1979).

The zinc content of crustaceans has been studied less than that of mollusks. The zinc content of several whole decapod crustaceans is rather consistent and ranged from 0.02 to 0.05 mg/g (National Research Council 1979).

Different groups have tried to ascertain the factors influencing the uptake of metals into fish tissues and have proposed several agents including food, metal levels in the water, and total environment exposure. However, a few of the more recent investigations have reported that the concentration of metals in fish is not dependent on any one factor but results from complex interactions of many factors (Cowx 1982). In laboratory experiments, the brown bullhead (Ictalurus) accumulated zinc 65 rapidly for the first 7 hours of exposure followed by a reduced accumulation rate. Gill and viscera attained the highest zinc concentration of the tissue analyzed.

Matthiessen and Brafield (1977) were cited by Hughes and Tort (1985) for work relating to the marked loss of zinc to fish soon after zinc treatment. They observed a greater loss (from 100 percent to about 40 percent) between 0 and 4 hours after transfer and about 40 percent remains in the fish or is lost very slowly. Versar (1979) determined that zinc exposed fish which were transferred to fresh water lost one-half their accumulation of zinc after 6 days followed thereafter by a much reduced rate of zinc elimination. Fish, decapod crustaceans, and certain species of polychaete worms appear able to regulate tissue concentration of zinc (National Research Council 1979).

According to Roch et.al.(1985) the mean concentration of zinc in rainbow trout muscle significantly correlated with the zinc concentration in the water ($P<0.05$, $r=.798$). In mussels, scallops, and freshwater bivalve mollusks, zinc is strongly localized in certain organs, whereas in oysters the metal is rather uniformly distributed throughout all tissues. Cowx (1982)

determined that metals were not evenly distributed throughout bodies of fishes; each element had a different distribution. High levels of zinc were found in the scales of both trout and char and also the kidney of the trout. These data on tissue distribution of metals are similar to observations made by others.

In a study done by Cowx (1982), no correlation was found between the concentration of Zn, Fe, Mn, and Cu in the tissues of trout and char and the length, weight, or age of these fish. Although similar findings have been reported elsewhere, both positive and negative correlations between the tissue metal concentration and growth parameters have been observed.

Cowx (1982) cites studies by Goodyear and Boyd (1972), Cross et al. (1973), and Weiner and Giesy (1977) where they proved that fish homeostatically control essential trace elements such as zinc.

Zinc is required in trace amounts by all algae for normal physiological and biochemical processes (Rai et. al.1980). Accumulation of zinc in aquatic plants was found to be directly related to the amount in the ambient water. The log of the zinc concentration in Chlorella cells was proportional to the log of the zinc concentration in the medium over a hundredfold range in the concentration of zinc. Halving the concentration of Zinc 65 available to Chlorella resulted in a 50 percent decrease in uptake and total amount accumulated. Previously, other investigations had reported similar results with certain other algae (National Research Council 1979).

The presence of other cations in waters containing zinc has been shown to reduce the amount of zinc taken up by aquatic plants. Zinc 65 uptake in the green algae, Golenkinia, was reduced when the concentrations of calcium, magnesium, potassium, and sodium were elevated (National Research Council 1979). Rai et. al. (1980) found that calcium at 20 mg/l had maximum decreasing effect on the toxicity of test metals on Chlorella vulgaris.

Beeby and Eaves (1983), studied the effects of zinc on the garden snail, Helix aspersa. The digestive gland was the main site of heavy metal accumulation as was confirmed by other individuals. Dry weight and age were the major factors governing the concentrations of zinc. Trace metal turnover is more rapid in the youngest snails, although the three age classes achieved broadly the same zinc concentration at the end of the experiment. This suggested that the final concentration of zinc is under physiological control.

In a study of zinc in the Newport River estuary in North Carolina, workers found three polycheate worms (Glycera americana, Diopatra cuprea, and Amphirite ornata) showed similar concentrations of metals regardless of large differences in metal concentration of the sediments in which the worms were burrowing and feeding. They suggested that these species may regulate body concentration of trace metals. Another group also found that zinc appeared to be regulated independently of the zinc level in the sediment. It was subsequently found that zinc in Neresis varied by a factor of only 2.7 although they were living in sediments where zinc concentration varied by a factor of about 30 (National Research Council 1979). In experiments to induce toxicosis, worms from sediments high in zinc were more resistant to zinc than were normal worms. This adaptation of Nereis was ascribed to a reduced permeability to zinc and more effective excretion in work done by Bryan and Hummerstone (1973) as cited by the National Research Council (1979).

12.3.2 Sampling of Aquatic Life

As described in Chapter 9, fish were collected from Flathead Lake by setting bottom gill-nets. Fishing of the nets yielded Dolly Varden, bull trout and whitefish. Fish tissue were analyzed for trace metals and PAH.

Zinc, copper, chromium, and selenium were found in the viscera and muscle tissue of Dolly Varden and lake whitefish collected from Kalispell Bay. The concentrations of the detected

metals were almost always higher in the viscera than in the muscle tissue. Very low levels of arsenic were found only in some viscera samples. Concentrations of chromium in viscera ranged from below detection limits (BDL) to 0.39 ug/g and from BDL to 0.57 ug/g. Copper concentrations in the viscera ranged from 0.17 to 27.0 ug/g and from 0.53 to 1.0 ug/g in muscle tissue. For selenium, concentrations ranged from 0.31 to 0.98 ug/g in the viscera and from 0.20 to 0.34 ug/g in muscle tissue. Zinc was by far the metal of highest concentration in the fish tissues with viscera concentrations ranging from 46 to 140 ug/g and muscle levels ranging from 3.4 to 9.0 ug/g.

Based upon the sediment and water concentrations in Flathead Lake the body burdens of the above metals are not unusual and are not at levels considered harmful for consumption. Copper and zinc, moreover, are both classified as essential trace elements for human nutrition.

Concentrations of zinc in the muscle tissue compare favorable with those found in lake char (Salvelinus namaycush) from Tadenac Lake, Ontario, Canada which ranged from 2.4 to 9.4 ug/g (Wren et al. 1983). Sediment concentrations of zinc in Tadenac were higher than in Flathead Lake ranging from 133 to 484 ug/g.

Concentrations of PAH above instrument detection limits (0.5 ug/g) were not found in any of the fish tissues analyzed.

12.4 Waterfowl Uptake of PAH

The slough area is provides a habitat for a variety of waterfowl. The following sections describe the studies conducted to determine the potential impact of the elevated zinc and PAH to waterfowl.

12.4.1 Literature Data on PAH Uptake by Waterfowl

Many studies have been conducted on both the acute (short-term) and chronic (long-term) effects of crude and refined oils on waterfowl. Many studies also have been conducted in regards to ingestion of oil and oil products by waterfowl. In studies

with mallard ducks (Anas platyrhynchos), Lawler et al. (1978) demonstrated accumulation of two and three-ring aromatic hydrocarbons into tissues from crude oil administered orally. The skin and underlying fat tissue accumulated aromatic hydrocarbons greater than the liver, breast muscle, heart muscle, brain, and blood. Furthermore, aromatic hydrocarbons did not accumulate in the same relative concentrations as found in crude oil suggesting differential uptake in metabolism.

Szaro et al. (1978) found that mallard ducklings fed 0.025, 0.25, 2.5, and 5.0 percent crude oil in the feed had evidence of liver and spleen pathology. In addition, behavior of ducklings was affected, as measured by avoidance responses, at the 2.5 and 5 percent oil diets. At even lower levels of oil diet (0.025 and 0.25 percent), plasma enzymes were elevated which indicated kidney and liver functions that are indicative of impending histopathology.

Hoffman and Gay (1981) studied the embryotoxic effects of BaP, chrysene and 7,12-dimethylbenz(a)anthracene in mallard ducks. These studies were conducted by externally applying PAH to the shell of an egg, and showed that PAH at relatively small levels are toxic to mallard embryos.

Belisle et al. (1981) found that both aliphatic and aromatic compounds (including naphthalene, methylnaphthalenes, fluorene, and phenanthrene) were found to accumulate in mallard eggs when the female diet contained 25,000 parts per million (ppm) petroleum. However, the residue levels of the aliphatic compounds were greater than those of aromatics.

Patton and Dieter (1980) studied the effects of petroleum hydrocarbons on hepatic (liver) functions of adult mallard ducks. One group of ducks was fed an ad libitum diet of breeder mash containing a 1 percent mixture of 0.96 percent (9600 ppm) paraffin and a 0.04 percent (400 ppm) aromatic hydrocarbon mixture. A second group was fed a 1 percent mixture of 0.60 percent (6,000 ppm paraffin) and 0.40 percent (4,000 ppm) aromatic hydrocarbons in the breeder mash. The aromatic hydrocarbons in the mash

included acenaphthene, acenaphthylene, and phenanthrene. Body weight decreased in the groups fed aromatic hydrocarbons during the first two months but returned to pretreatment levels after five months of the study. This was thought to be a result of decreased food intake due to the noxious odor of the food. No mortality, no gross organ abnormality, or decrement in liver functional capacity was shown. These parameters indicated that petroleum hydrocarbons produced little change in hepatic cells.

This data (Patton and Dieter 1980) suggested that the adult mallard is able to tolerate high concentrations of petroleum hydrocarbons for an extended period of time. The liver function test results suggested a dose-response relationship with the level of aromatic hydrocarbons in the feed, and though an increased physiological demand on the adult ducks was evident, the duck was able to tolerate high concentrations of these aromatic hydrocarbons for extended periods of time.

The only known study of direct uptake of PAH by waterfowl from food is that of Tarshis and Rattner (1982). In this study, crayfish, which were artificially contaminated with 14C-naphthalene, were force-fed to one year redhead ducks to determine accumulation of the 14C-naphthalene. The crayfish contained approximately 45 ppm (mg/kg) naphthalene wet weight providing a daily dose of 1.25 mg/bird. Greatest concentrations of 14C-naphthalene were found in the fat, gallbladder, and bile and kidney tissues. While there was appreciable accumulation of 14C-naphthalene, the exposure did not cause any overt signs of toxicity in the duck.

12.4.2 Potential for PAH Uptake by Waterfowl at Somers

There are data published that demonstrate uptake of PAH into waterfowl by ingestion of PAH-contaminated feed and one study demonstrates uptake of PAH through the food chain directly. Based on rough estimates of food intake, mallard ducklings in the studies would only have to ingest between 15 and 150 mg of oil a day to demonstrate pathological or physiological disfunction

(Szaro et al. 1978). Furthermore, if ducklings matured in areas with chronic low level oil pollution, this magnitude of oil could either be directly ingested or indirectly obtained through the food chain. On the other hand, petroleum hydrocarbons have not been unequivocally demonstrated to be present in tissues of seawater-adapted birds that have consumed petroleum-contaminated food (Holmes et al. 1978).

In evaluating the potential for PAH uptake from the food chain, a series of theoretical extrapolations were made both from the published literature and the measured sediment and water PAH concentrations in the Somers, Montana slough. Phase I results (ERT 1985) indicated total PAH in the slough at 329 ng/l (nanograms per liter or parts per trillion). Subsequent measurements of PAH in the slough water during the Phase II RI indicted no detectable PAH at a 1 ug/l detection limit (see Section 5.1 of this RI). PAH in sediments were found to be 342 mg/kg from slough sample No. 3 (See Section 5.2). Using these values, theoretical PAH levels potentially accumulated in waterfowl can be calculated. It must be noted that these calculations assume no metabolism in the waterfowl and are probably worst-case theoretical values. Theoretical waterfowl uptake of PAH from food sources contaminated via water exposure and uptake (329 ng/l in water) is calculated as follows.

The bioconcentration factor (BCF) for aquatic snails in the literature (Lu et al. 1977) is 4,860. Multiplying 329 ng/l PAH in water by 4,860 (BCF) results in an accumulation of 1.6 mg/kg of PAH (ppm) in snails. Assuming that a one-half pound (0.23 kg) goldeneye duckling might consume as much as 30 percent of its body weight per day (Karesh 1986), then 0.07 kg of benthos might be consumed daily (0.23 kg x 0.3). Then multiplying 0.07 kg of benthos times the calculated 1.6 mg/kg of PAH in snails, then approximately 0.11 mg of PAH would be consumed daily by goldeneye ducklings.

Szaro et al (1978) stated that pathological changes occurred in mallard ducklings fed on estimated 15 mg of crude oil per day.

Using Patton and Dieter's (1980) estimate that 15 percent of crude oil (South Louisiana crude) is PAH then the estimated dose for pathological changes in mallard ducklings is 2.25 mg PAH per day per duck (0.15 x 15 mg/day). The calculated dose of PAH (0.11 mg/day) is more than 20 times less than that estimated by Szaro et al. (1978) (2.25 mg/day). Therefore, there appears to be little risk of pathology damage to ducklings consuming food organisms contaminated by PAH in the water from the Somers slough.

Theoretical waterfowl uptake of PAH from food sources contaminated via sediment (342 mg/kg in sediment) exposure and uptake is calculated as follows: The estimated BCF for marine polychaete worms in the literature (Lyes 1979 as cited by Neff 1985) is 4.1 for the stomach wall of this marine worm. Assuming that freshwater worms (Oligochaetes) might have a similar BCF, then an evaluation may be made. Assuming 342 mg/kg of PAH in sediment times the estimated BCF of 4.1, then approximately 1,402 mg of PAH might be contained in a kg of worm tissue. However, the mean lethal concentration of 7 PAH compounds to marine polychaete worms is 1.15 mg/l (ppm) (Neff 1979). Therefore, it is unlikely that worms would accumulate body burdens greater than the lethal levels estimated in the literature. Using this 1.15 ppm concentration, an estimate of potential uptake by waterfowl can be made.

Assuming that a one-half pound (0.23 kg) goldeneye duckling might consume as much as 30 percent of its body weight per day of benthic worms, then 0.07 kg of worms (0.23 kg x 0.3) might be consumed daily. Multiplying 0.07 kg of worms by the calculated 1.15 mg of PAH per kg of worms results in approximately 0.08 mg of PAH per duckling per day that could theoretically be consumed. This daily dose (0.08 mg PAH) is almost 30 times less than the estimated dose of 2.25 mg PAH (0.15 x 15 mg/day) per day per duckling which produced pathological changes (Szaro's et al. 1978). The evidence indicates that there probably would be no physiological impairment or pathology to ducklings from this

estimated daily dose of PAH.

Based on the literature, accumulation of PAH in waterfowl tissues over a lifetime might occur. Tarshis and Rattner (1982) demonstrated accumulation of naphthalene as a result of consumption of freshwater crayfish contaminated with this 14C-naphthalene in the laboratory. However, the levels of naphthalene in crayfish were much higher (45 ppm) than those estimated in this evaluation. The study was conducted for only three days and the potential for accumulation at much lower doses over long periods of time are as yet unknown. Factors such as metabolism of parent PAH compounds in birds are known to exist (Jellinck and Smith 1973) and Lee et al. (1985). Complicating factors also include the unknown rates of accumulation from food of PAH compounds other than naphthalene in waterfowl. Naphthalene is known to be one of the more accumulative compounds (Neff 1979), and may not be indicative of accumulation of other PAH compounds.

In summary, it appears that PAH compounds may be accumulated at much lower levels than have been shown to produce pathological and physiological changes in laboratory feeding studies. Due to the extremely low PAH exposure levels and factors such as waterfowl migration and feeding habits, it seems unlikely that waterfowl inhabitating the slough adjacent to the site would suffer any physiological or pathological change.

12.5 Zinc Uptake by Waterfowl

12.5.1 Literature Review

Di Giulio and Scanlon (1984), did a study on 15 species of ducks obtained from the Chesapeake Bay area to determine the relationship between food habits and tissue concentrations of heavy metals. Of the 807 ducks examined, a total of 774 livers, 266 kidneys, and 271 ulnar bones were obtained for metal analyses from ducks killed between the 1976-77 and 1979-80 wintering seasons, inclusively. Mallards had zinc levels of 118 ppm in ulnar bones, 161 ppm in livers, and 86 ppm in kidneys. Gadwalls had 130 ppm in ulnar bones, 197 ppm in livers, and 94 ppm in

kidneys. Pintails had 140 ppm in ulnar bones, 122 ppm in livers, and 74 ppm in kidneys. Green-winged teals had 105 ppm in livers. No relationships were apparent between food habits and tissue concentrations of zinc. Among the elements analyzed, zinc concentrations displayed the least variability and had the most normal distributions.

Di Giulio and Scanlon (1984), reviewed a study of zinc toxicity in mallards done by Gasaway and Buss (1972) where the observed mean liver and kidney concentrations in controls of 162 ppm and 115 ppm , respectively. These are similar to values observed for mallards in their Chesapeake Bay study. Zinc induced toxicosis due to a dietary zinc supplementation of 3,000 ppm (wet weight) was associated with mean liver and kidney zinc concentrations of 1,200 ppm and 1,760 ppm, respectively.

A total of 301 specimens of sixteen different species of waterfowl were collected from the Ouse Washes, England in research done by Parslow, Thomas, and Williams (1982) to obtain estimates of the background levels of the metals from a relatively unpolluted area. The mean liver concentrations of zinc for gadwalls, mallards, pintails, and teals were 113.0 ppm, 157.2 ppm, 159.9 ppm, and 148.0 ppm, respectively. These values are generally in good agreement with the Chesapeake Bay study results.

As reported by the National Research Council (1979), Gasaway and Buss (1972) found that mallard ducks fed high levels of zinc suffered severe paralysis; some ducks were unable to walk after 30 days. The lowest level fed them was 3,000 ppm, but even at this level food consumption decreased. At higher levels, consumption was reduced to the point that none of the groups received as much zinc in a 30-day period as did those on the 3,000 ppm intake. The high mortality in the groups receiving over 3,000 ppm zinc may have been caused primarily by decreased feed intake.

Poultry are able to tolerate levels of 1000 to 2000 ppm without adverse effects as cited by Goede (1985) in a study by

Ewan (1978). The National Research Council (1979) sites work done with poultry where the addition of 3,000 ppm zinc to a corn-soybean meal ration significantly reduced growth in chickens. Only a slight reduction occurred in growth with 2,000 ppm, and taking the chicks off the high zinc diets brought about weight gains during the next 6 weeks equal to that of the controls. Mortality from Salmonella gallinarium was not affected by 2,000 ppm zinc in the diet. Growth in turkey poults was reduced slightly by 4,000 ppm but not by 2,000 ppm.

One of the most complete studies was done by Beyer et. al. (1985), on wildlife living near two zinc smelters operating since about 1900 in the Palmerton, Pennsylvania area. Two study sites were selected; Palmerton and Bake Oven Knob. The authors report that soils, microflora, vegetation, lichens and arthropods inhabiting soil litter have all been affected by the smelters.

Amphibians, birds, and small mammals were included in the study as well as wildlife food items, such as plants and invertebrates. The soils involved are strongly acidic (pH 5.0 to 5.9). Of the two sites studied, the Palmerton site was the most heavily contaminated containing up to 24,000 mg/kg zinc in soils. The maximum zinc in soils at the Bake Oven site was 960 mg/kg. A comparison of the concentrations of zinc (ppm) in the foliage, acorns and berries, fungi and invertebrates at both of these sites are as follows:

	Palmerton	Bake Oven Knob
Foliage	660 ppm	148 ppm
Acorns/Berries	59 ppm	27 ppm
Fungi	320 ppm	120 ppm
Moths	250-540 ppm	140-340 ppm
Caterpillars	280 ppm	170 ppm
Cicadas	220 ppm	270 ppm
Bark Beetle Larvae	1450 ppm	470 ppm
Beetles	150 ppm	120 ppm

No gross or microscopic lesions attributable to metal poisoning were found in tissues of 15 birds collected from Palmerton and 12 from Bake Oven Knob. Furthermore, the zinc

concentrations in the songbirds at Palmerton were only 17 percent higher than those at Bake Oven Knob.

The Palmerton site provides an opportunity to study the effects of metals on wildlife. If high concentrations of zinc in soil are a potential hazard to wildlife, one would expect to find evidence of it at Palmerton. The results of this study indicate that only a small proportion of the zinc in the Palmerton soil became incorporated into plant foliage and that even less became incorporated into acorns and berries. Also, the concentrations in the foliage bore little relation to the degree of contamination of the soil. The authors generalized that those species that decompose organic matter in soil and their predators are most likely to have high zinc concentrations. High zinc concentrations in soil are not necessarily hazardous to most of the wildlife species at the site, but to those species whose food chains are dependent on soil organisms. Animals feeding on foliage, berries, fungi, or herbivorous insects have less exposure through their food chains.

12.5.2 Potential for Zinc Uptake by Waterfowl at Somers

In evaluating the potential for zinc uptake from the food chain, a series of theoretical extrapolations were made from both the published literature and the measured sediment and water zinc concentrations in the slough. The zinc concentration in the water of the slough was treported to be a maximum of 0.091 mg/l (ppm). The sediments of the slough were also tested and the zinc concentrations were found to be 1,800, 4,300, and 4,100 mg/kg in the Phase II samples and over 8,000 mg/kg in a Phase III sample.

According to Bartonek and Hickey (1969), 57 percent of the food consumed by juvenile redheads is algae (Chara). The bioconcentration factor (BCF) for freshwater algae (Versar 1979) is 4,000. Multiplying 0.091 ppm zinc in the slough water by the BCF of 4,000 we get 364 ppm zinc in the algae. The remaining 43 percent of the juvenile redheads's diet is of animal origin. The BCF for freshwater invertebrates in the literature (Versar 1979)

is 40,000. Multiplying 0.091 ppm zinc in the slough water by 40,000 results in 3,640 ppm zinc in invertebrates. The juvenile redhead would therefore be exposed to 0.57 x 364 ppm zinc in algae + 0.43 x 3640 ppm zinc in animals or 1780 ppm zinc in its total daily diet.

According to Munroe (1939), a summary of food percentages of 441 species of Goldeneye from various localities indicated that 26.09 percent of their diet is plant and 73.91 percent is animal. The BCF for freshwater plants is 4,000 and the zinc concentration of the water is 0.091 mg/l so the plants contain approximately 364 mg/kg zinc. Then, 0.26 times 364 mg/kg equals 95 ppm of zinc in plant material. Taking the BCF for freshwater invertebrates of 40,000 and multiplying this by the zinc content of the slough water (0.091 ppm) the zinc concentration of the invertebrates is 3,640 mg/kg. The dietary zinc from animal foods is 0.74 x 3640 ppm = 2700 ppm. Therefore the total dietary exposure of zinc would be 95 ppm plus 2700 ppm or 2795 ppm.

The above cases all have findings of dietary zinc concentrations from less than 2000 ppm to 2795 ppm. Literature data indicates adverse impacts to mallard ducks fed high levels of zinc. They were reported to suffer severe paralysis; some ducks were unable to walk after 30 days. The lowest level fed to the ducks in this study was 3,000 ppm, but even at this level food consumption decreased. The addition of 3,000 ppm zinc to a corn-soybean meal was reported to significantly reduced growth in chickens.

The literature data therefore indicted a potential risk to waterfowl from zinc exposure. This resulted in the design and execution of a program for observing waterfowl behavior at the site slough with provisions for sampling young-of-year waterfowl, if required.

12.5.3 Waterfowl Observation and Sampling

Section 9 provides the details of the waterfowl survey and sampling effort conducted during this RI. Observations were made

comparing the overall use of the two sloughs (i.e. species types and densities), as well as feeding habits and clinical symptoms of any adverse conditions (i.e. listlessness, ruffled feathers, ataxia, etc.) in adult and young ducks. The types of behavior evaluated are the broad classifications taken from the detailed Observation Data Sheets included in Appendix L. Parameters on the observation data sheets were marked as normal if no abnormal manifestations were observed. Environmental observations were included as a part of the study including water levels, predation, location of nests, and vegetation.

The nest search revealed that many more birds were nesting on the Somers site slough than on the control slough. Coots were by far the most prolific nester, particularly on the Somers Site slough. Successful hatching occurred in almost every nest that had not been predated. Fifty eight percent of the coot nests were successful on the site slough. Fifty five percent of the coot nests were successful on the control slough. Most of the terrestrial nesters were predated upon at both locations.

On the control slough, the predominant species were the coot and redhead and on the site slough the coots were by far the dominant species with goldeneye being the predominant duckling type. Over subsequent observation periods, coots and redheads were the predominate species seen. Goldeneye and coot ducklings were the most abundant species found at both locations. Other species observed were mallards, ruddy ducks, pied billed grebes, blue-winged teals, shovelers, gadwall and pintail ducks.

Observations of adult waterfowl involved mostly feeding, preening, and loafing behavior, but some sexual and mating behavior was observed as well. Ducklings were most often seen feeding, swimming, and sleeping and behaving normally. Overall, all observable appearance and behavior were normal. Fledging was found to be normal in all species observed over the time period. The only abnormal behavior observed was one blue-winged teal which quacked almost continually during the observation period of August 8, 1987 on the site slough. It continued to swim with

other teal and feed the entire time, however.

Goldeneye ducklings of about the same age were obtained from each slough. Goldeneyes feed by diving and they ingest aquatic insects and crustaceans, which represents the greatest bioconcentration of zinc in foodstuffs. A routine postmortem examination was performed on each duckling. The posting of ducks did not reveal any abnormalities. There were no pathological lesions found on any of the killed ducklings and all ducklings were found to have abundant internal and external body fat and full digestive organs which indicated that they were eating well and in good physical health.

The liver, kidneys, and muscles were collected for laboratory analysis. Laboratory results indicated that the liver concentrations of zinc from ducks from the site slough ranged from 35 to 37 mg/kg and from 38 to 40 mg/kg from ducks killed on the control slough. Kidney results were 18 to 25 mg/kg on the site slough and 23 to 24 mg/kg on the control slough. Muscle results were 18 to 24 mg/kg on the site slough and 25 to 29 mg/kg from birds from the control slough.

These laboratory results showed that tissue zinc concentrations were very comparable between sloughs. When nesting success, hatchability, and results from tissue zinc analysis are considered along with detailed observations by qualified persons over a five month time period, a finding of no adverse impact is fully justifiable.

13. RISK-BASED CLEAN-UP GOALS FOR SOMERS

Using the risk assessment approach described in Section 11, it is possible to back calculate levels of constituents in soils, water and air that could be present and results in "acceptable" levels of exposure. In developing these goals, no consideration will be made of the technical feasibility of achieving a goal.

For compounds that are handled as noncarcinogens, the acceptable daily intake values for chronic exposures will be used to determine acceptable concentrations in the environment. For compounds that are handled as carcinogens, carcinogenic potency factors will be used to derive environmental concentrations which correspond to various risk levels. The exposure scenarios developed in Section 11 will be used.

13.1 Noncarcinogenic Compounds

Acceptable intake values have been estimated for noncarcinogenic PAH compounds (see Tables 11-5 and 11-6). Published values exist for phenol, a compound which was found to be present in the groundwater wells downgradient of the CERCLA Lagoon, and for zinc. Because of the margin of safety built into the acceptable intake values, exceedance of the number has no immediate meaning with respect to specific health effects or the frequency or magnitude of the health effects. However, the clean-up goals for Somers will be based on a non-exceedance of those values.

Table 13-1 presents the acceptable intake values and the calculated air, water and soil concentrations. Development of soil quality goals assumed the site could be used for residential purposes. Therefore, the maximum exposure rate which occurs from ages 1 to 6 (body weight = 15 kg and soil ingestion = 100 mg/day) was used in the analysis. Use of the naphthalene acceptable intake estimate of 0.0053 mg/kg/day provides the most stringent environmental control. This value as a clean-up goals results in air quality goals of 18.6 ug/m3, water quality goals of 186 ug/l

TABLE 13-1

ASSESSMENT OF CLEAN-UP GOALS FOR NONCARCINOGENIC COMPOUNDS

Parameter	Acceptable Daily Intake (mg/kg/day)	Acceptable Concentration in		
		Air (ug/m3)	Water (ug/l)	Soil (mg/kg)
Naphthalene	0.053	186	1,855	6,760
Naphthalene	0.0053	18.6	186	67,600
Acenaphthalene	0.03	105	1,050	38,260
Acenaphthene	0.20	700	7,000	255,000
Fluorene	0.07	245	2,450	89,250
Phenanthrene	0.07	245	2,450	89,250
Anthracene	0.07	245	2,450	89,250
Fluoranthene	0.07	245	2,450	89,250
Pyrene	0.06	210	2,100	76,500
Phenol	0.04	140	1,400	51,000
Zinc	0.21	735	7,350	56,350

---

* Value assumed equal to average AIC for other noncarcinogenic PAH

Air exposure assumes inhalation of 20 m3 of air/day and 70 kg body weight.

Water exposure assumes injestion of 2 liters of water/day and 70 kg body weight.

Soil exposure assumes childhood residential exposure at 1 to 6 years of age for 7 months per year and a 20 percent matrix. No matrix effect assumed for zinc.

and soil quality goals of 6,760 mg/kg. Acenaphthene has the highest acceptable daily intake at 0.2 mg/kg/day. Use of this value results in acceptable air, water and soil concentrations of 700 ug/m^3, 7.0 mg/l, and 255,000 mg/kg, respectively. It is therefore recommended that the lower bound naphthalene acceptable intake value be used to guide the clean-up of noncarcinogenic compounds. This will ensure no adverse health effects from other noncarcinogenic compounds as they will be reduced to concentrations much less than their acceptable levels through the control of naphthalene. This is especially true at the Somers site where naphthalene is often the prevalent PAH compound detected. It is also expected that phenol and zinc concentrations will be reduced to less than their acceptable concentrations (each at more than 50,000 mg/kg in soil) by control of naphthalene concentrations.

13.2 Carcinogenic Compounds

EPA has published a potency factor for only one of the known or suspected carcinogenic PAH, benzo(a)pyrene. This value has since been "withdrawn" by the agency because of problems with the method used to derive the number. EPA is currently reviewing a new report (Clements 1987b) in which a revised potency factor for benzo(a)pyrene has been derived. Although this value is still considered draft and is under EPA review, it is expected to eventually replace the original EPA estimate.

In estimating the risks associated with other carcinogenic PAH compounds, the general practice is to utilize B(a)P as a surrogate for the other compounds. It is well recognized that this will overestimate the risks because B(a)P is one of the most potent chemicals among PAHs. Several groups (Chu and Chen 1984; Clements 1987a,b) have been working toward developing relative potency factors for other PAH. Data from the Clements work was presented in Table 11-6.

Target risk levels of 10^{-4} to 10^{-7} have been considered for the Somers clean-up goals. Table 13-2 presents the calculated "acceptable" soil and water concentrations using 1) the original

TABLE 13-2
ASSESSMENT OF CLEAN-UP GOALS FOR CARCINOGENIC COMPOUNDS

Parameter	CPF (mg/kg/day)-1	Relative Potency (Clement)	Acceptable Water Conc. (ng/l) at a risk level of 1x10-4	1x10-5	1x10-6	1x10-7
Benzo(a)pyrene (EPA)	11.5		304	30.4	3.0	0.3
Benzo(a)pyrene (Clement)	3.22	1.0000	1,087	109	10.9	1.1
Benzo(a)anthracene	11.5	0.1450	2,099	210	21.0	2.1
Chrysene	11.5	0.0044	69,170	6,917	692	69.2
Benzo(b)fluoranthene	11.5	0.1400	2,174	217	21.7	2.2
Benzo(k)fluoranthene	11.5	0.0660	4,611	461	46.1	4.6
Indeno(123-cd)pyrene	11.5	0.2320	1,312	131	13.1	1.3
Dibenzo(ah)anthracene	11.5	1.1100	274	27	2.7	0.27
Benzo(ghi)perylene	11.5	0.0100	30,435	3,043	304	30.4

Parameter	CPF (mg/kg/day)-1	Relative Potency (Clement)	Acceptable Soil Conc. (mg/kg) at a risk level of 1x10-4	1x10-5	1x10-6	1x10-7
Benzo(a)pyrene (EPA)	11.5		92	9.2	0.9	0.09
Benzo(a)pyrene (Clement)	3.22	1.0000	328	32.8	3.3	0.33
Benzo(a)anthracene	11.5	0.1450	634	63.4	6.3	0.6
Chrysene	11.5	0.0044	20,909	2091	209.1	20.9
Benzo(b)fluoranthene	11.5	0.1400	657	65.7	6.6	0.7
Benzo(k)fluoranthene	11.5	0.0660	1,394	139.4	13.9	1.4
Indeno(123-cd)pyrene	11.5	0.2320	397	39.7	4.0	0.4
Dibenzo(ah)anthracene	11.5	1.1100	83	8.3	0.8	0.08
Benzo(ghi)perylene	11.5	0.0100	32,800	3280	328.0	32.8

Water exposure assumes injestion of 2 liters of water/day and 70 kg body weight.

Soil exposure assumes lifetime residential exposure 7 months per year with a 20 percent matrix effect.

EPA estimated carcinogenic potency factor for benzo(a)pyrene, 2) using the recently revised benzo(a)pyrene value developed by Clements, and 3) the using the original EPA estimate in conjunction with the Clements relative potency factors for other carcinogenic PAH.

Calculated goals in water and soil based on benzo(a)pyrene having a carcinogenic potency factor of 11.5 mg/kg/day^{-1} are as follows:

	10^{-4}	10^{-5}	10^{-6}	10^{-7}
Water, ng/l	304	30.4	3.0	0.3
Soil, mg/kg	92	9.2	0.9	0.09

Use of the draft revised benzo(a)pyrene carcinogenic potency factor of 3.22 mg/kg/day^{-1} increases these concentrations by a factor of about 3.5. Table 13.2 also presents the calculated levels of other carcinogenic PAH using their relative potencies and the original EPA carcinogenic potency factor of 11.5. For one compound, dibenzo(ah)anthracene, the calculated acceptable levels are reduced as since this compound is believed to be more potent than benzo(a)pyrene. The remaining acceptable concentration for other carcinogenic PAH compounds are reduced by factors ranging from about 4.3 for indeno(123-cd)pyrene to 227 for chrysene.

At this point, recommendation of a single risk level for the clean-up goals at Somers will be deferred. It is recommended that the Feasibility Study consider the range of risk levels and evaluate alternatives in terms of their ability to achieve the various risk level goals. It is recommended however that the relative potencies of the various PAH be considered in these subsequent evaluations. Although no official EPA documents has come out in support of these relative potency factors, they are believed to represent the soundest scientific data currently available. Many conservative assumptions have been made throughout the Risk Assessment, particularly the assumption that

the site will be developed for residential purposes. Although this is not beyond the realm of possibilities, it is much more likely that the future use of the site will be of an industrial or commercial nature. The "acceptable" soil concentrations would decrease significantly with the use of this more realistic land use scenario.